A CLINICAL STUDY OF ULCERS OF THE FOOT

A dissertation submitted to the TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,CHENNAI -10.

in partial fulfillment of the requirements for the degree of the

M. S. (GENERAL SURGERY)

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI.

2013

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"A CLINICAL STUDY OF ULCERS OF THE FOOT"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. V.PANDY** M. S., Professor, Department of Surgery, TIRUNELVELI MEDICAL COLLEGE HOSPITAL, Tirunelveli Medical College, Tirunelveli.

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ACKNOWLEDGEMENT

So many people have contributed to the successful completion of this dissertation that I cannot list them all and must ask the forgiveness of those whom I omit. This study required the cooperation of many people. My sincere acknowledgements to all the participants in this study of mine.

With the most sincere and deepest sense of gratitude, I thank **Dr.S. SOUNDARAJAN M.S**., Professor and Head, Department of General Surgery, Tirunelveli Medical College Hospital, Tirunelveli Medical College, for his patience, constant encouragement, guidance and constructive criticism. His valuable suggestions and timely advice were of immense help to me throughout all phases of this study.

I extend my sincere thanks to **Dr. V. PANDY,** M.S.,Professor, Department of General Surgery, Tirunelveli Medical College, Tirunelveli, for his valuable guidance, encouragement and suggestion during this dissertation.

I express my heartfelt gratitude to **Prof. Dr. MAHESHWARI**, **Prof. Dr. M.S. VARADARAJAN**, **Prof. Dr. ALEX EDWARDS**, **Assistant Professors** and all the teaching faculties for their timely advice and encouragement in preparing this dissertation.

I thank **Dr.S. Senthil Arumugam M.S.**, **Dr. Amalan M.S.**, **Dr.B. M. Pabitha Devi M.S., Dr.G.Nirmal Kumar M.S., Dr. Irene Aruna Edwin**

M.S.,,for helping me to complete this study without whose help the study could have not been completed.

I sincerely acknowledge the help and assistance rendered by my fellow postgraduates, junior postgraduates, CRRI's, and SOT staff. Last but not the least; I thank all the patients for their kind co-operation in carrying out the study successfully.

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LIST OF ABBREVIATIONS USED

TAO	→	Thromboangiitis Obliterans
HLA	→	Human Leucocyte Antigen
ESR	→	Erythrocyte Sedimentation Rate
TC	→	Total Count
DC	→	Differential Count
LDL	→	Low-density Lipoprotein
VDRL	→	Veneral Diseases Research Laboratory
RBC	→	Red Blood Corpuscles
WBC	→	White Blood Corpuscles

ABSTRACT

BACKGROUND

A chronic ulcer of the foot is a frequent condition, with prevalence in the population over 60 years of age. The incidence of ulcers is rising as a result of the ageing population and increased risk factors for atherosclerotic occlusion such as smoking, obesity and diabetes.

Ulcers can be defined as ‘break in the continuity of the covering epithelium either skin or mucous membrane due to molecular death’. In general, the slow healing tendency is not simply explained by depth and size, but caused by an underlying pathogenetic factor that needs to be removed to induce healing.

The main causes are varicose veins, lower extremity arterial disease and diabetes, less frequent conditions are infection, vasculitis, skin malignancies and ulcerating skin diseases such as pyoderma gangrenosum. For a proper treatment of patients with foot ulcers it is important to be aware of the large differential diagnosis of foot ulceration and to effectively manage the conditions

AIMS AND OBJECTIVES

• To compare and analyze the distribution of age, sex, systemic disease and location of the ulcer among 100 cases of the study group.

• To study the clinical features of various types of foot ulcers.

• To study the usefulness of applied investigations.

• To identify the methodology to effectively manage the condition.

• To identify the steps to prevent as far as possible foot ulcers in high-risk individuals prone to the condition.

METHODS

Prospective study of 100 cases of chronic foot ulcers admitted at Tirunelveli medical college Hospital, Tirunelveli, during the period March 2011 to March 2012, with regular dressing, debridement, treating the underlying systemic disease, skin grafting and amputation were done.

RESULTS

In a study group of 100 cases, most of the patients with leg ulcers had an underlined systemic disease such as diabetes mellitus, varicose veins, arterial occlusion secondary to atherosclerosis, leprosy and malignancy.

KEYWORDS

Chronic non-healing ulcer; Diabetic leg and foot ulcer; varicose ulcer; Tropic ulcer; Arterial ulcer; Malignant ulcer.

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INTRODUCTION

Chronic ulcers of the lowerlimb is frequent condition and wide in distribution they may be associated with a number of Medical, Surgical & Dermatological condition,most commonly dealt with and treated in the surgical wards and OPD.

The incidence of ulcers is more in aging population and increased risk factor for atherosclerotic occlusion such as smoking, obesity and Diabetics.

Ulcer can be defined as ‘break in the continuity of the covering epithelium either skin or mucous membrane due to molecular death’, In general the slow healing tendency is not simply explained by depth and size. But caused by a underlying pathologic fact that needs to be removed to induce healing.

The problems of lower limb ulcer represent a wide spectrum of etiology, pathology, severity and morbidity. The main causes are varicose veins, lower extremity arterial disease and diabetes. Less frequent conditions are infections, vasculitis, skin malignancies and ulcerating skin diseases such as pyoderma gangrenosum. But even rare condition exists such as recently discovered combination of vasculities and hypercoagulability.

For a proper treatment of patients with lower limb ulcers, it is important to be aware of the large differential diagnosis and different causess of foot ulcers. The causes may be various but the anatomical situation of ulcers in the foot by itself can give rise to problems that can at times test the ingenuity and patience of the surgeons

Various studies have been conducted and a number of procedures and techniques have evolved with varying degree of success. It is common to see patients with different types of ulcers due to various etiology and underlying systemic diseases. Moreover, foot ulcers form a good bulk of patients in our hospital. Treatment of these ulcers forms a challenging task as well.

I have therefore in my present study attempted to analyze the ulcers of the foot.

This study comprises of:

Review of literature with regard to historical aspects, etiology, anatomy, pathology, pathophysiology, clinical features and diagnosis of chronic foot ulcers along with the various modern investigative studies required for the diagnosis.

Clinical investigation and histopathological study of patients of chronic foot ulcers are done in patients who are admitted in Tirunelveli medical College Hospital, Tirunelveli.

AIMS AND OBJECTIVES

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REVIEW OF LITERATURE

I. HISTORICAL ASPECT OF LEG ULCERS

The incidence of chronic foot ulcer is as old as history, as with any disease of mankind. The most common and noted ulceration of the chronic foot ulcers for many years is Stasis ulcers.Hippocrates, the legendary father of medicine himself had a leg ulcer. He was against treating various ulcerations by surgical means. He treated multiple varicose veins by puncturing them at different levels to avoid non-healing of ulcers and about 400 years B.C. He wrote - " In case of an ulcer, it is not expedient to stand, especially if the ulcer be situated on the leg" (Sarkar P. K. Ballantynes).**1**

In 4th Century B.C, healing by faith was practiced as seen in clay models, which are taken from Temples of Athens.

Avicenna (982 - 1027 AD) gave a good account of diabetes and was the first one to describe diabetic gangrene.

Mollers Christenses during the period 1250 - 1500 A.D reported changes in the long bones of the leg due to leprosy. He found these changes in the skeletons in burial grounds of Lazar Hospital. Earlier, pathological evidences to date are in Mummies of 2nd century B.C. Sir Benjamin Collin Brodie (1789 - 1862) was the first to notice and describe the reverse flow of

blood by means of a clinical test. Half a century later, this test was termed as "Trendlenberg's test".

In 1828 Marjolin described the carcinomatous ulcers originating from degenerative burns scars - this ulcers bears his name.

Celsus, the Roman physician & Hippocrates described treatment by bandaging for chronic ulcers in the leg and related them to varicose veins. John Hunter (1878) attempted to explain these ulcers in terms of venous stasis due to the pressure of the column of blood while in upright posture.

Gay & Spencer's in 1868 wrote 2 important books on venous ulcers, which stressed the role of deep vein thrombosis and other lesions of arteries and veins (both superficial and deep) in etiology of lower limb ulcerations. Gay also described perforating ankle veins and suggested use of the term venous ulceration (M. Wayne Flye).**2**

Linton drew attention to incompetence of communicating calf veins as potential cause of venous ulceration (Linton RR).**3**

Pryce (1887) described the association of ulceration and vascular diseases in diabetes.

From the 10th to 18th centuries various physicians including **Halu, Abbas, Avicenna, Falopio** and **Pare** attributed ulceration of the leg to accumulation of black bile or bad tumours and believed that ulceration of the

leg served useful purpose in getting rid of these live Substances (Shami S. K. Shield).**4**

In 1909 Burger described a syndrome of vascular occlusion in which arteries, veins, and nerves of the extremities were involved in extensive fibrosis that resulted in ulceration.

Although a minor traumatic incident is often the immediate cause of the ulcer, the underlying pathology is usually vascular. Callam et al (1985) estimated that 70 percent of leg ulcers are venous in origin, 10 per cent are arterial and 10-15 per cent are of mixed arterial/venous origin. More unusual causes include malignancy, vasculitis, neuropathy, metabolic disorders such as pyoderma gangrenosum, and ulcers associated with disorder such as sickle cell ulceration (Moffatt and Harper 1997)

II. **INCIDENCE AND OCCURRENCE OF FOOT ULCERS**

Accurate data concerning the incidence of non-fatal diseases are difficult to obtain and statistics are usually derived from hospital attendance records and general practice surveys. In under developed countries, the incidence of ulcer may be greatly under estimated and apparent differences between populations may be affected by differences in age structures.

The site of ulceration is recorded using the method of Callum; 90% of the ulcers were present in the gaiter area, 2 % in the foot and 8% in the leg (Baker S. R. et al).**6**.

A basic study of 4422 healthy working adults aged between 20 and 70 years in Europe resulted in detection of chronic venous insufficiency in 19% of men and 25% of women. Other surveys conducted in Europe and America came out with similar results and surveys confirmed the increased incidence of ulcerations in females.

Information from other parts of the world other than Europe and America is sparse. Schulz, Finaly and Scott (1962), in Transvaal, found that 0.1% of 2000 consecutive Bantu patients attending surgical outpatient department had venous ulcers, but do not give figures for the incidence of non-venous leg ulcers, while Dogliotti (1970) in Johannesburg found the incidence of leg ulcers (unspecified) in Bantu patients referred to hospital to be 1.75%. Finally and Park (1969) compared the incidence of varicose eczema in their middle aged Indian patients (7%) with that in comparable "white" patients (3%) and Bantu (1%) but give no figures for the incidence of venous ulcerations.

III. **ANATOMY OF THE LEG AND FOOT**

The leg is that part of the lower limb below the knee and the terminal part of the leg below the ankle is the foot.

Front of the leg and dorsum of foot

Superficial fascia of the front of the leg and the dorsum of the foot contains the following parts:

- Superficial veins.
- Cutaneous Nerves
- Lymphatics
- Small unnamed arteries.

The subcutaneous bony surfaces are not covered by the deep fascia of the leg but are attached to it at its borders. It is thick in the upper part of the leg and gives origin to the underlying muscles while in the lower part is thin and forms retinacula around the ankle.

Intermuscular septa are formed in the deep fascia, which tends to divide the leg into compartments. The anterior and the posterior intermuscular septa are attached anterior and posterior borders of the fibula and divide the leg into anterior, lateral and posterior compartments. In the posterior compartment a superficial transverse facial septum separate tibialis posterior from the long flexors of the toe.

Muscles of the anterior compartment are – Tibialis anterior, Extensor halucis longus, Extensor digitorum longus and Peroneous tertius.

Anterior Tibial Artery

The main artery that is found in the anterior compartment of the leg is the anterior tibial artery. The perforating branch of the peroneal artery feeds this artery. Anterior tibial artery is the smaller terminal branch of popliteal artery.

Surface Anatomy

In surface projection of the anterior tibial artery begins 2.5 cm below the medial side of fibula and ends at the midpoint between the malleoli. The vessel can be felt pulsating lateral to the tendon of extensor halucis longus at the ankle.

Course

The originating point of the anterior tibial artery is at the back of the leg at the lower border of the popliteus muscle and it penetrates the anterior compartment of the leg through an opening in the upper part of the interosseous membrane.

In the anterior compartment it runs vertically downwards to a point midway between the two malleoli, where it ends by becoming continuous as the dorsalis pedis artery. Anterior tibial artery is accompanied by two venae – commitantes and the deep peroneal nerve is lateral to it in the upper and lower third and anterior to it in the middle 1/3rd of the leg.

Variations

The perforating branches of the peroneal artery and the perforating branches of the posterior tibial artery may partially or completely replace the anterior tibial artery.

Fig 1: Posterior tibial, popliteal and lateral plantar artery

Fig 2: Anterior tibial and Dorsalis pedis artery

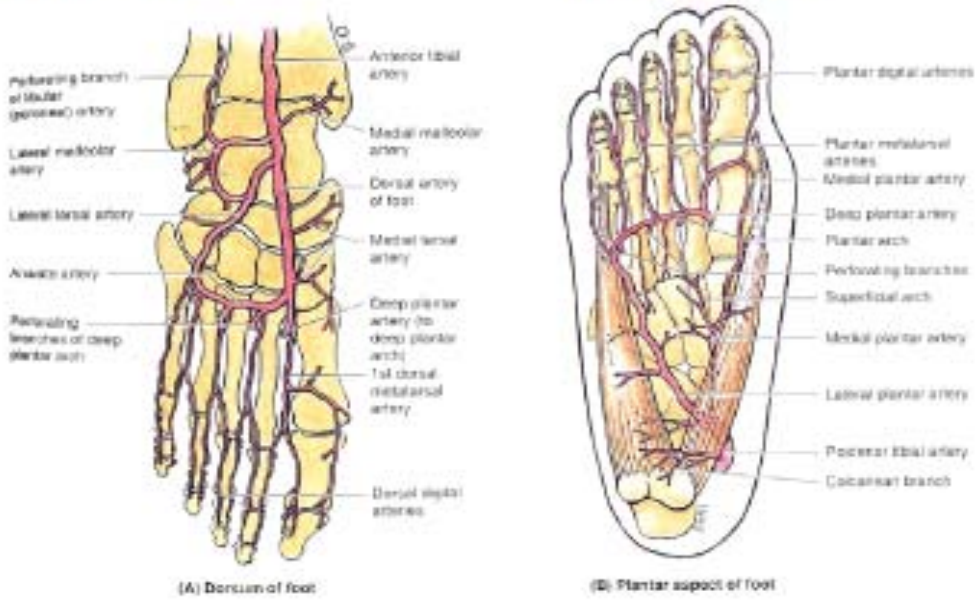


Fig 3: Arteries of the foot

Branches

a) The adjacent muscles are supplied by the muscular branches.

b) Anastomotic branches feed the knee and the ankle. It is formed by:

- ❖ Anterior medial mallelor branch of anterior tibial.

- ❖ Medial tarsal branches of dorsalis pedis.
- ❖ Medial malleolar branches of posterior tibial.
- ❖ Calcaneus branches of posterior tibial and
- ❖ Twigs from the lateral plantar artery.

Lateral malleolar network lies just below the lateral malleolus. It is formed by:

- ❖ Anterior lateral malleolar branch of anterior tibial.
- ❖ Lateral branches of dorsalis pedis.
- ❖ Perforating branch of peroneal artery.
- ❖ Twigs from the lateral plantar artery.

Dorsalis Pedis Artery

The direct continuum of the anterior tibial artery, which forms the chief artery of the dorsum of the foot, is the dorsalis pedis artery.

Surface anatomy

Being superficial the vessel can be felt pulsating along a line from the midpoint between the malleoli to the proximal end of the first intermetatarsal space.

Course

The originating point of this artery is in front of the ankle between the two malleoli. The artery lies along the medial side of the dorsum of the foot and reaches the proximal end of the first intermetatarsal space. It deeps

downwards between the two heads of the first dorsal interosseous muscle and ends in the side by completing the plantar arterial arch.

Relations

a) Superficial:

- Skin and fascia with interior and exterior retinaculum.
- Extensor hallucis brevis, crosses artery from lateral to medial side.

b) Deep:

- Capsular ligament of ankle joint
- Talus, navicular and intermediate cuneiform

c) Medial:

- Extensor hallucis longus

d) Lateral:

- First tendon of extensor digitorum longus
- Medial terminal branch of deep peroneal artery.

Branches

1. Lateral tarsal artery supplies extensor digitorum brevis, tarsal joints and ends in lateral malleolar network.
2. Medial tarsal branches joints medial malleolar network.
3. Arcuate artery is a large branch that arises opposite the medial cuneiform bone. It gives off second, third and fourth dorsal metatarsal

artery each of which divides into dorsal digital branches for the adjoining toes.

4. First dorsal metatarsal artery gives a branch to medial side of big toe and divides digital branches for adjacent sides of first and second toes.

Cutaneous Innervation

1. Saphenous nerve which is a branch of the posterior division of femoral nerve pierces the deep fascia an the medial side of the knee, runs downwards in front of great saphenous vein and supplies skin of the medial side of the leg and medial border of the foot up to the ball of big toe.
2. Infrapatellar branch of saphenous nerve pierces the sartorius and deep fascia on the medial side of the knee and supplies the skin over the ligamentum patellae.
3. Lateral cutaneous nerve of calf, which is a branch of the common peroneal nerve, supplies skin of their upper 2/3rd of lateral side of leg.
4. Superficial peroneal nerve is a branch of common peroneal nerve on the lateral side of the neck of fibula. It pierces the deep fascia at the junction of the upper 2/3rd and the lower 1/3rd of the lateral side of the leg and supplies skin over the lower 1/3rd of the lateral side of the leg and the dorsum of the foot.

5. Sural nerve is a branch of the tibial nerve given of in the popliteal fossa. It pierces the deep fascia in the middle of the back of the leg accompanies the small saphenous vein and supplies the skin of the lower ½ of the back of the leg and lateral border of the foot upto the tip of the little toe.
6. Deep peroneal nerve supplies the cleft and adjacent borders of the first and second toe.
7. Digital branches of the medial and lateral plantar nerve supply the distal parts of the toes.

Deep Peroneal Nerve

This is the nerve of the anterior compartment of the leg and the dorsum of the foot. It is one of the two terminal branches of peroneal nerve.

Course

Originating on the lateral side of the neck of the fibula, it enters the anterior compartment by piercing through the anterior intermuscular septum from where it accompanies the anterior tibial artery in to the leg. It is the main supplier to the anterior muscle compartment. The nerve ends on the dorsum of the foot close to the ankle joint by dividing into lateral and medial terminal branches. The extensor digitorum brevis, tarsal joints and the second dorsal interosseous muscles are supplied by the lateral terminal branch. The medial

terminal branch ends by supplying the first interdigital cleft, proximal joint of the big toe and often the first dorsal interosseous muscle.

Branches

1. The adjacent sides of the first and the second toes are supplied by the cutaneous branches.
2. The muscles of the anterior compartment of the leg and the extensor digitorum brevis on the dorsum of the foot are supplied to by the muscular branches.
3. Articular branches supply the ankle joint, tarsal joint, tarsometatarsal and metatarsophalangeal joint of the big toes.

Lateral Side of the Leg

The lateral compartment of the leg is a bound unit. There are many muscular membranes involved in binding this compartment, they are:

- ❖ Anteriorly by anterior intramuscular septum
- ❖ Posteriorly by posterior intramuscular septum
- ❖ Medially by lateral surface of fibula.
- ❖ Laterally by deep fascia of the leg.

Contents

Muscles

- ❖ Peroneus longus
- ❖ Peroneus brevis.

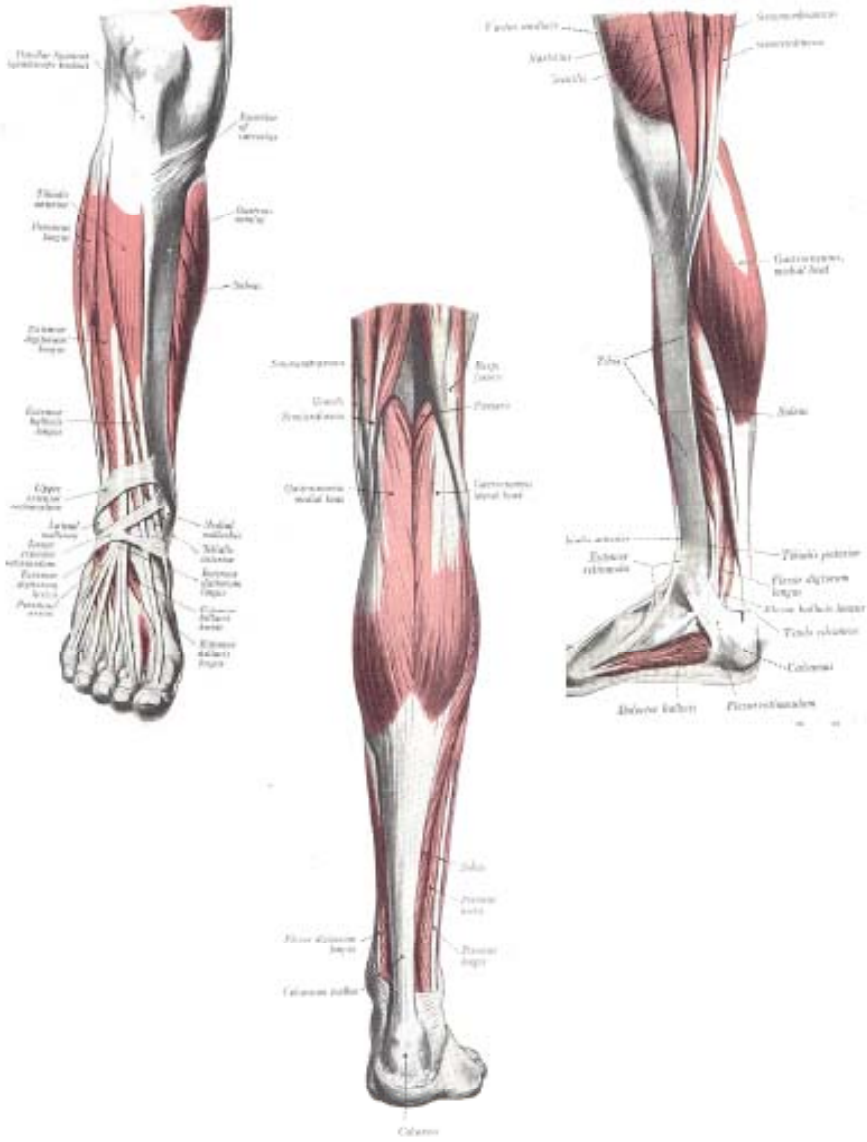


Fig 4: Muscles of the leg

Actions

- The peroneus longus and the brevis are evertors of the foot. The movements of inversion and eversion help to adapt the foot to uneven ground.
- Along with the other muscles present around the ankle the peroneal help to maintain the stability of the joint.

- ❖ The peroneus longus helps to maintain the lateral longitudinal arch of the foot.

Vessels

- ❖ Arterial supply is derived from the branches of peroneal artery; veins drain into short saphenous vein.

Nerve

- ❖ Superficial peroneal nerve

Superficial Peroneal Nerve

One of the common peroneal nerve's two terminal branches which is the main nerve to the lateral compartment of the leg is the superficial peroneal nerve.

Course and Relation

It begins on the lateral side of the neck fibula, descends through the substance of peroneus longus in the upper 1/3rd, lies between the peroneus longus and brevis muscle in the middle 1/3rd and then descends to a groove between the peroneus brevis and extensor digitorum longus. At the junction of the upper 2/3rd and lower 1/3rd of the leg it pierces deep fascia and divides into medial and lateral branches, which descend into the foot.

Branches and distribution

a) Muscular branches – To peroneus longus and brevis.

b) Cutaneous branches – through the terminal branches supplies 1/3 of the lateral side of the leg and the dorsum of the foot. The lateral branch supplies the third and the fourth clefts of the toes.

c) Communication branches – communication between the saphenous and the deep peroneal nerves takes through the medial branch and the lateral branch with the sural nerve.

Medial side of the leg

The medial surface of the shaft of the tibia is the originator of this part of the leg. A large part of the medial surface of the leg is covered only by the skin and the superficial fascia and hence this part is largely subcutaneous. The surface at the upper end provides anchorage to the tibial collateral ligament and also acts as the insertion point of the sartorius, gracilis and semitendinosus, all of which are covered by a thin layer of deep fascia. The great saphenous vein lies in the superficial fascia as they cross the lower 1/3 of this surface. This skin, fascia and the periosteum of this surface are supplied by saphenous nerve.

Rear of the leg

The hind leg contains the posterior compartment of the leg. This posterior compartment is bound by the following:

a) Anteriorly by the rear surface of the Tibia, Interosseous membrane, Fibula, Posterior intermuscular septum

b) Posteriorly by the deep fascia of the leg. Superficial fascia of the back of the leg contains:

- The small and the great saphenous vein
- Several cutaneous nerves
- Medial and lateral calcaneal arteries.

The muscles of the posterior compartment are divided into superficial and deep Group. The superficial muscles are the gastrocnemius, Soleus and the Plantaris. The deep muscles are the Popliteus, Flexor digitorum longus, Flexor hallucis longus and the Tibialis posterior.

Posterior Tibial Artery

This is the larger terminal branch of the popliteal artery.

Surface anatomy

The posterior tibial artery from the middle line of the calf at the level of neck of fibula to the midpoint between the medial malleolus and the prominence of the heal, where its pulsations can be felt.

Course

Originating at the lower border of the popliteus between the tibia and the fibula deep to gastrocnemius, it enters the back of the leg and passes deep into the soleus. It then runs downwards and medially to reach midway between the medial malleolus and medial tubercle of calcaneum to terminate

deep into the flexor retinaculum by dividing into a large lateral and a small medial plantar artery.

Branches

- Peroneal artery
- Muscular branches for the posterior compartment.
- Nutrient artery to tibia is the largest nutrient artery in the body.
- Anastomotic branches – Circumflex fibular for the knee,

  communicating branch

  for peroneal, medial malleolar and calcaneal artery for ankle and heel.
- Terminal branches – medial and lateral plantar.

Peroneal Artery

The lateral and the posterior compartments of the leg are supplied by the largest branch of the posterior tibial artery and are called the peroneal artery.

Course

Beginning just 2.5 cm below the lower border of the popliteus, it descends along the medial crest of the fibula. It is found in a fibrous canal between the tibialis and the flexor hallucis longus, it further continues downward behind the ankle joint and terminates into the lateral calcaneum branches.

Branches

1. Muscular branches to posterior and lateral branches.
2. Nutrient artery to the fibula.
3. Anastomotic branches.

- ❖ Perforating branch joins the lateral malleolar network.
- ❖ Communicating branch anastomosis with a similar branch from posterior tibia.
- ❖ Calcaneous branches join the lateral malleolar network.

Cutaneous Innervation

1. Saphenous nerve is a branch of posterior division of femoral nerve and supplies the skin of medial side of the leg and medial border of foot upto the ball of the big toe.
2. Posterior division of the medial cutaneous nerve of thigh supplies the skin of the uppermost part of medial area of calf.
3. Posterior cutaneous nerve of the thigh supplies the skin of the upper ½ of the central area of the calf muscle.
4. Sural nerve is a branch of the tibial nerve and supplies the skin of the lower half the central area of the leg, lower 1/3rd of the lateral area of the calf and also lateral border of the foot upto the tip of the little toe.

5. Lateral cutaneous nerve of calf is branch of the common peroneal nerve and supplies the skin of the upper 2/3rd of the lateral area of the leg both in front and behind.

Sole of the foot

The skin of the sole of the foot is thick, firmly adherent to the underlying plantar aponeurosis and is creased.

Cutaneous innervations

a) Medial calcaneal branches of tibial nerve supplies the skin of posterior and medial portions of the sole.
b) Branches from medial plantar nerve of the anteromedial portions with medial 3 ½ digits.
c) Branches from lateral plantar nerve supplies the anterolateral portions with 1 ½ lateral digits.

Superficial fascia

This is more fibrous and dense. The fibrous brands bind the skin to the deep fascia and divide the subcutaneous fat into small tight compartments. The fascia is very thick and dense over the weight bearing points.

Deep Fascia

Deep fascia of the sole forms:

a) Plantar aponeurosis in the sole.

b) Deep transverse metatarsal ligaments between the metatarsophalangeal joints.

c) Fibrous flexor sheaths in the toes.

Muscles of the first layer of the sole are Flexor digitorum brevis, Abductor hallucis and Abductor digitorum. The muscles of second layer are Flexor digitorum accessories and lumbricals, which are four in number. Muscles of third layer of sole are Flexor hallucis brevis, Adductor hallucis. And Flexor digiti minimum brevis. The fourth layer of the sole contains the muscles – the Dorsal interossei which are four in number and lie between metatarsal bones and the Plantar interossei which are three in number and lie below the metatarsal bones.

The main arteries of the sole are the medial plantar artery and the lateral plantar artery.

The main nerves of the sole are medial and lateral plantar nerves.

Venous drainage of the leg and foot

Superficial veins

These are the long and short saphenous systems and their tributaries. They lie in the superficial fascia. The presence of smooth muscles and fibrous tissue makes them thick walled. They contain valves, which are more in number at the distal part than proximally. A large portion of blood from the superficial veins is drained into the deep veins through perforators.

The Dorsal venous arch

This is formed by the four dorsal metatarsal veins, each of which is formed by two digital veins. It lies on the dorsum of the foot over proximal part of the metatarsal bones.

The long saphenous vein

This is formed by the union of medial end of dorsal veins are which medial marginal vein, from the medial side of the great toe. It passes upwards

Fig 5: Superficial and deep veins of the leg and foot

in front of the medial malleolus and runs along the medial border of the tibia to reach the back of the knee. From here it passes upwards and slightly anteriorly to reach the cribriform fascia and pierces it and drains into the femoral vein.

The short saphenous vein

This is formed by union of the lateral end of the dorsal venous arch with lateral marginal vein from the lateral side of the little toe. It passes upwards behind the lateral mallelous to reach the back of the leg and ascends first lateral to the tendoachilles and then along the midline of the calf to pierce the deep fascia of the popliteal fossa and opens into popliteal vein, It drains the lateral border of the foot, the heel and back of leg. Sometimes the whole short saphenous veins open into the long saphenous system through an accessory saphenous. Also occasionally it can either end in the long saphenous system just below the knee or in the deep muscular veins.

The deep veins

These are the anterior tibial, posterior tibial, peroneal, popliteal veins.

These veins accompany their corresponding arteries, supported by the surrounding powerful muscles. They also have valves, which are more numerous than in superficial veins. The muscular veins are also valved except for soleal vein. Deep veins are more efficient channels than superficial veins because of the driving force of the muscular contractions.

Perforating Veins

These are of two types:

a) The direct perforators – these connect the superficial veins to the deep veins. The long short saphenous veins are the large direct perforators. There are about five small perforators along the long saphenous vein and one perforator along the short saphenous vein.
b) The indirect perforators – these connect the superficial veins to the deep veins through muscular veins.

LYMPHATIC DRAINAGE

Superficial lymphatics

These form large trunks and are more numerous than the deep lymphatics. They run in the superficial fascia in two streams.

a) The main stream follows the long saphenous vein and ends in the lower vertical group of the superficial inguinal lymphnodes.
b) The accessory stream follows the short saphenous vein and ends in the popliteal lymph nodes.

Deep Lymphatics

These are fewer than superficial lymphatics and drain all structures lying deep to the deep fascia. They terminate mostly into the deep inguinal nodes either directly or indirectly through popliteal nodes.

Fig 6: Layers of the skin

a. Stratum germinitivum

b. Stratum spinosum

c. Stratum granulosum

d. Stratum lucidum

e. Stratum corneum

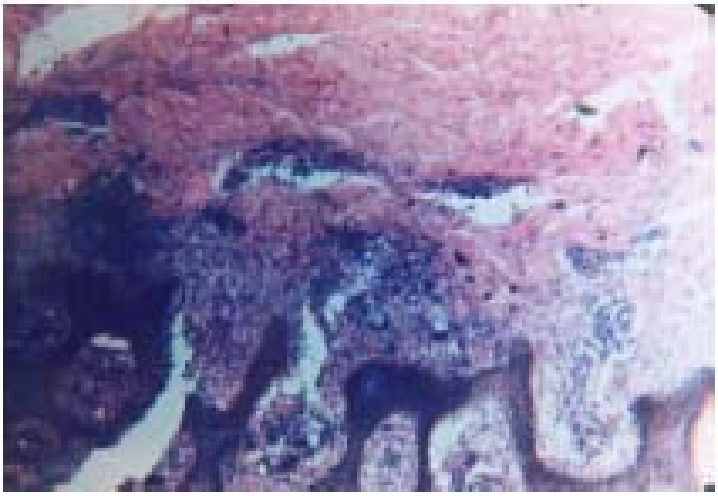


FIG 7 :Squamous cell carcinoma – high power field

IV. HISTOLOGY OF THE SKIN

The skin is anatomically composed of three distinct strata and they are named as (From the outermost to the inner layer). Epidermis, Dermis and Subcutaneous fat.

The Skin

Its development

The ectoderm and the mesoderm act as the originator of all the constituents of the skin. Or to put it in simple terms the skin is developmentally derived from the ectoderm and the mesoderm.

The Epidermis

The epidermis is composed mostly of keratinocytes, and stratified squamous epithelium. The epidermis is made – up of the five layers.

The stratum corneum is the first layer and this is thickest in palmar and plantar areas. The second layer is stratum lucidium which is an even, colourless, translucent and a shining band just below stratum corneum. The third layer is stratum granulosum. The forth layer is stratum malphigii (Stratum spinosum) which consists of row of mosaic like keratinocytes. The last and the fifth layer of the epidermis is the stratum germinatum (Basal layer). This layer consists of rectangular columnar cells arranged vertically over a wavy basement line.

Cell differentiation in Keratinocytes takes place in the direction of the external environment and they yield a cornified surface membrane. The process of differentiation whereby germinative keratinocytes move outward to become fully cornified cells takes approximately 14 days and are present in transit through the cornified layer. The replacement of the epidermis and in very thick epidermis, in the second layer as well. In the basal layer 3-5 % of the cells synthesize DNA at any given time. During upward migration towards maturation, keratinocytes undergo characteristics changes. Basal, spinous, granular and cornified layers are the strata that reflect stages in the conversion of a germinative keratinocyte from the beginning to the end and produce the epidermal differentiation.

In the upper malphigian layer dendritic cells resembling monocytes are found and are called as Langerhan's cells.

Melanin pigment is synthesized and secreted by melanocytes that are dendritic cells. The melanocytes are present all over the body but their distribution is not even. The intensity of coloration is further determined by the total number of cells and intercellular distribution of melanin granules within the epidermal keratinocytes.

The Dermis

The dermis or the corium is a dense fibrous or elastic layer, which provides the strength and elasticity to the skin. Composed primarily of white

fibrous tissue,some yellow elastic fibres and cement like substances called the cellular elements. Blood vessels, glandular structures, lymph vessels, hair follicles, muscle elements prolongations of fatty tissue and nerves with terminal organs of touch and sensation are all contained and supported in this layer. The dermis is covered over its entire surfaces with numerous conical papillae. This stratum is called ‘The papillary layer’, whereas the deep stratum is termed ‘The reticular layers’. The combined anatomical unit of papillary and reticular dermis is called ‘The adventitial dermis’.

The level of intimacy between the papillary dermis and the epidermis is easily visible in their alterations as a result of inflammatory skin diseases. They form a morphological and a functional unit, which work together.

Subcutaneous Fat

Subcutaneous fat, like the dermis is derived from mesenchyme and mesenchymal cells. These cells give rise to lipocytes (fat cells) as well as fibrocytes. The lipocytic cells manufacture so much fat inside themselves that in the process they tend to flatten the nucleus against the wall of the cell. Strands of collagen divide the population of the fat cells into lobules. These fibrous elements known as trabeculae have major vascular networks, lymphatics and nerves and are analogous to the adventitial dermis. The subcutaneous fat varies in thickness from one are to another. Functionally the

subcutaneous fat acts as an insulator, shock absorber and a chronic reserve depot.

Nerves

Sensations such as touch, pressure, temperature, pain and itch are received by millions of microscopic dermal nerve endings. They are most numerous on hairless parts like palms, soles of the feet, fingers and clitoris, the erogenous zones. These tiny end organs terminate principally in the papillary dermis and around hair follicles. These nerve endings can be free or encapsulated, myelinated or unmyelinated and most of these are visualized with difficulty using the heamotoxylin and eosin stain.

V. BIOLOGICAL PROCESS OF HEALING

In the wound healing process three distinct phases can be visualized.

1. The phase of inflammation (Day 1-4)
2. The phase of proliferation (Day 5-20)
3. The phase of differentiation (Day 20 onwards).

The phase of inflammation

A wound causes a change in the tension prevalent in the tissue and this causes a change in the charge, in the collagen molecule. Contract of the collagen with the blood flowing in the wound causes the activation of kinin and complement cascade, which is the initial phase in the clotting process. A haemostatic plug is formed by the adherent platelets. The blood vessels

undergo brief construction followed by vasodilatation under the influence of histamines from the platelets and mast cells. An increase in the capillary permeability is noted. Serotonins, the kinins, and the prostaglandins maintain capillary engorgement. White and red blood cells escaping from the walls of the blood vessels form a network of fibrins over the wound site, which within three hours is surrounded by a few lymphocytes and an increasing number of polymorphs. A distinctive lytic behaviour is observed on part of these neutrophils because of their lysosome content. By the fifth day monocytes would have become the dominant cell type because they are observed in maximum number. They are phagocytic and ingest cellular debris. Reduction or delay in this macrophage function will delay wound healing. By the end of the first phase new capillaries bud from the endothelial cells in the capillaries near the wound edge, while in connective tissue around the vessels mesenchymal cells differentiate to become fibroblasts. Clinically in this phase the classical features observed that are manifested by the wound are heat, redness, tenderness, swelling and loss of function.

The phase of Proliferation

By the fifth day fibroblasts have began to synthesize collagen and Ground substance.

Collagen

Collagen is the extra cellular fibrous framework that gives strength and form to the tissue. Dependent on the amino acid sequence there are various types of collagen. Proline, hydroxyproline and glycine are the most predominant types of collagen that are seen. The types of collagen that is found in the skin, bone, tendon and ligament is the type 1 collagen, type 2 collagen occurs in the cartilage and the type 3 collagen occurs in the fetal dermis and is replaced by type 1 collagen at the time of birth. Granulation tissue gives rise to the type 1 collagen. Hydroxylation of immature protocollagen requires oxygen, ferrous ions and ascorbic acid. Further maturation involving glycosylation produces more stable tropocollagen. Tropocollagen is held together by weak electrostatic forces and is soluble in weak salt solutions. This collagen, though stable, is not inert, and it undergoes constant turnover under the influence of tissue collagens. Thicker collagen fibers soon abound and are laid down haphazardly.

Ground Substance:

The Ground substance is an amorphous matrix of connective tissue and is basically constituted of water, electrolytes, mucopolysaccharides and glycoprotiens. It is a thin gel, but in cartilage it is elastic. It is produced by fibroblasts and is involved in the formation and maturation of extra cellular collagen. It exists as a water rich phase and in equilibrium with a colloid

phase. Complexes occurring between the proteins and the polysaccharides provide unique property to the ground substance. The types of mucopolysaccharides include chondoritin, chondroitin-4 sulphate, chondroitin-6 sulphate, dermatan sulphate, keratin sulphate and hyaluronic acid.

The phase of differentiation

There is no clear demarcation between proliferation and the differentiation phase. The later starts in the proliferating granulation tissue and continues indefinitely. There is a rationalization of copious new blood vessels and notably a remodeling of the haphazard arrangement of the collagen fibers. The synthesis of the new collagen is in a more orderly fashion along the lines of tension in the scar. Collagen turnover and remodeling in the scar never stops. Indeed, the turnover of the collagen in the scar tissue is faster than in other tissues.

The epithelial defect in the incised wound is initially plugged with fibrin collagen and the epidermis turns downwards over the edge of the underlying dermis. After 24 hours, basal cells are mobilized on the underlying surface of the epidermis and by about 48 hours the advancing epithelial edge would have undergone cellular hypertrophy and mitosis. The epithelium migrates but stops when it meets the opposite advancing epithelium.

When there is superficial skin loss, the dermal pits that have been left behind act as islands for regenerating epithelium. However, once lost there is no regeneration of hair follicles, sweat or sebaceous glands in the new epidermis.

Healing by first intension

The healing of wounds caused by accident, assault, warfare and surgical operations has always been a central, consideration in surgical practice because any breach in the surface of the body – the skin and mucous membrane – exposes the deeper tissue to the danger of infection. Therefore, it is necessary to assist the healing process of the body to restore an intact surface as soon as possible. Immediate closure of a wound (Primary suture) using sutures, clips and adhesive materials favour healing with minimal scarring and is called healing by first intension.

Healing by second intension

When the wound edges do not come together or when there is irreparable skin loss or when the wound becomes infected and breaks apart and has to be laid open, then in such cases healing takes place by second intension. In this type of healing, the wound is healed by a filling of granulation tissue. This process of healing is far slower than wound healing by first intension and there is more amount of scar tissue left behind (Ulcers usually heal in a similar fashion).

Other methods of providing skin cover – in the presence of devitalized tissue, swelling tension and skin loss – include delayed primary suture, skin grafting and second suture.

Factors contributing to rate of wound healing

There are many factors, which govern the rate at which a wound heals. Some of these factors are discussed below:

- **Blood Supply**

Wounds incorporated on the face and hands may seem horrifying initially but they tend to heal well because of good blood supply to these organs. Injuries sustained in areas of relatively less vascular supply such as areas below the knees, over the shin tend to take a much longer time to heal.

- **Tension**

Tension of the tissue inhibits the local blood supply and leads to wound failure.Local swelling and therefore tension builds up automatically during the first 48 hours after the injury as part of the phenomenon of inflammation. Haematoma, venous stasis (Eg. In a dependent limb) and infection also increase tension.

- **Age**

The age factor plays an important role in the wound healing process. The most important factor in the wound healing process is the protein

turnover rate. As the age increases this capacity of the body to turnover proteins is reduced considerably leading to slower wound healing rates.

- **Rests**

Granulation tissue has a delicate blood supply that is easily damaged by movement and shearing forces. Rest only to the part of the body with ulcer is indicated such as POP cast or slab.

- **Infection**

Once infection is established the fibroblasts must compete with the bacteria and the inflammatory cells for oxygen and nutrients. Thus, overall collagen synthesis is inevitably reduced and the collagen breakdown is enhanced by collagenolytic enzyme activity. Infections are a major factor in wound healing failure.

- **Malnutrition**

Defective synthesis of both collagen and ground substance can be directly linked with malnutrition. Severe protein calorie malnutrition has long been implicated in the failure of wounds to heal, while lesser degrees of malnutrition depress healing as well. Vitamin C is necessary for the synthesis of ground substance. Vitamin D is essential for new bone formation and vitamin A for epithelialisation. Calcium, zinc, copper and manganese are essential minerals. In patients with burns and intestinal fistulas, in particular, become zinc depleted and require supplementation.

- **Uremia**

Experimentally the addition of urea to tissue cultures of fibroblasts inhibits their growth. Clinically uremia is implicated in the retardation of wound healing in patients with renal failure.

- **Jaundice**

Jaundice is associated with reduction in wound strength. The appearance of fibroblasts and the formation of new blood vessels are both delayed. Bioposies of the skin in jaundice patients show a reduction of the enzyme proline – hydroxylase involved in the collagen maturation.

- **Steroids**

An inflammatory response is essential for wound healing to proceed normally.Steroids depress wound healing by their anti-inflammatory action. New vessels are abnormal and sparse, as are fibroblasts. If steroids are given after the inflammatory phase of wound healing there is little increase in overall effect of healing.

- **Radiation**

Radiation causes cell death by damaging both DNA and disrupting intracellular metabolism.

VI. AETIOLOGY AND PATHOLOGY

About 95% of the leg ulcers are due to vascular aetiology (Gilliland)**7** and venous ulcers account for up to 90% of cases (Burton. S. Claude)**8**,

(Callam MJ, et al)**9**. Arterial disease accounts for 5-10%; most of the others are due to neuropathy, usually diabetic or a combination of these diseases (Yound JR).**10** Diabetic ulcers are common on the toe and the heel (Hansson Carita).**11**. Arterial insufficiency and / or diabetes may also be the causatives for ulcers below the line of the shoe. Ulcers at the ankles in the gaiter zone and venous ulcers are mostly caused by varicose veins (Hansson Carita). **11** Primary varicosity of the long saphenous system and / or short saphenous system is the causative factors for venous ulcerations (Hoare MC et al).**13**

The elevated ambulatory pressure in peripheral venous system in venous insufficiency manifests itself not only in form of disturbed microcirculation but also and particularly in microangiopathic changes. These include decrease in capillaries, glomerulus like changes and decrease in oxygen content. (Junger, M Stiens. A).**14** It has been also noted that perivascular fibrin cuffs and skin hypoxia precede lipodermatosclerosis in limbs at increased risk of developing a venous ulcer (Stacey M. C., Burnand K.G et al).**15**

Classification of foot ulcers

a. Venous ulcerations
b. Arterial insufficiency – Thromboangiitis Obliterans, atherosclerosis, Raynaud's embolic occlusions (sub acute bacterial endocarditis).
c. Diabetic neuropathic ulcerations.

d. Neoplastic ulcers – epithelioma melanoma, basal cell carcinoma and malignant change in long standing scars (Marjolin's ulcer).
e. Tropical ulcers including leishmaniasis, fungal infections.
f. Specific infections – tuberculosis, syphilis and AIDS.
g. Blood dyscrasias – severe anemia, sickle cell anemia, thalassemia, hereditary

   spherocytosis and leukaemia.
h. Nutritional and metabolic disturbances.
i. Skin sensitivity or allergy.
j. Trauma.
k. Necrosis by injection of chemicals, insect bites, snakebites or radiation.
l. Repeated trauma.
m. Rheumatoid arthritis.
n. Systemic autoimmune and micro vascular diseases.**16**

There are various stages in the evolution of the ulcers. They are:

- **The extension stage**: Floor of the wound is covered in slough and the base is indurated.
- **The transition stage**: separation of the slough combined with the clearing of the floor of the wound and appearance of red granulation tissue marks this stage.

- **The healing stage**: transition of the granulation tissue into fibrous tissue is observed, which later contracts and the wound healing by epithelisation is seen particularly in this stage.

The Venous Ulcer

Histopathologic reasons caused by venous insufficiency, is one of the main causes of venous ulceration. Changes taking place in the cluster of thick walled capillaries within a thickened fibrotic papillary dermis, accompanied by siderophages and extravasated erythrocytes in variable numbers also contribute to the overall situation of ulceration. Occurrence of perivascular fibrin cuffs in the skin is seen in venous insufficient areas. They are often present in the walls of superficial vessels and evident as smudgy eosinophilic areas. (fibrin cuff theory). Lipodermatosclerosis or sclerosing panniculitis demonstrates fibrosis and thickening of the subcutaneous septa. An early lymphocytic infiltrate of the subcutaneous septa is gradually replaced by a mixed infiltrate with increasing fibroplasia and sclerosis. In the late Lipodermatosclerosis there is minimal inflammation and subcutaneous sclerosis.

Dysfunction of the calf muscle pump can result in venous insufficiency in the deep, connecting or superficial veins; arteriovenous fistulae, or muscle dysfunction as a result of fibrosis, neuropathy or inflammatory diseases. Valvular damage caused due to thrombosis may lead

to deep venous insufficiency. Venous ulcers are mainly attributed to be caused by venous insufficiency; multisystem incompetence of the valves is also a common cause.

Another plausible hypothesis suggests that reduction in the blood flow rate and endothelial damage allows white cells to form plugs, which in turn plug the capillaries. These cells release inflammatory mediators increasing vascular permeability. This causes tissue ischemia and ulceration (white cell trapping theory).

Arterial Ulcers

Arterial or ischaemic ulcers are most commonly due to atherosclerosis and hence encountered in older adults. They can be also seen in younger adults and here usually peripheral arterial disease like Thromboangiitis Obliterans is the cause. The other rare causes of ischaemic ulcers are Raynaud's diseases and phenomenon.

Atherosclerosis develops at twice the frequency in patients who smoke compared with non-smokers (Coffman J. D.)**17** about 50% of the patients have lipoproteinemia.Patients with diabetes develop the disease at an earlier age than non-diabetics and have more severe and progressive disease. The distribution of disease is also different in that diabetics have less aorto-illiac disease and more disease of vessels between knees and ankles.

There is focal accumulation of lipids, mucopolysaccharides, blood and blood products, fibrous tissue and calcium deposits in the intima of the arteries. Localized areas of thickening of the intima by fibroblastic proliferations and phagocytes laden with lipid materials are seen. The media becomes atrophic with thin strands of muscles, disrupted elastic lamella, collagen tissue and calcium deposits. Enlarging plaques encroach upon the lumen despite dilatation of the artery, and plaques may ulcerate. Hemorrhages occur within the arterial wall. Thrombi are formed and finally occlude the vessel lumen. Another important feature here which causes sudden gangrene with ulceration is atheromatous embolisation. It is due to embolisation of small pieces of atheromatous plaques and debris to the arteries of extremities. This is called as blue toe or trash foot syndrome.

Another important arterial disease, which can cause ulceration and gangrene, is Thromboangiitis Obliterans (Buerger's disease).

The etiology of TAO remains unknown. Almost all patients who develop this disease are smokers and the syndrome sometimes abates following cessation of smoking. An increased frequency of HLA-A9 and HLA-B8/B5 have been reported but not found by all investigators (Millis J. L. et al).**18** The pathogenesis involves production of ischaemia and all its manifestations by an inflammatory action of medium and small arteries of extremities and also obstruction by thrombi.

In acute stage there is panvasculitis of the arteries and veins; the diagnostic finding being arterial thrombi with foci of microabscess and giant cells. In chronic stage only fibrotic obliteration of arteries is seen and diagnosis can only be surmised since atherosclerosis is absent.

Diabetic Ulcers

The various factors, which are the contributions for the cause of ulcers, are Hyperglycemia, microangiopathy, Neuropathy, Liability to infection and Alteration in blood flow.

The precipitating factors are injuries and infection. Injuries could be physical injuries like penetrating wounds, disruption of skin and injury due to continuous localized blunt pressure

In a diabetic condition of either type 1 or type 2, hyperglycemia may lead to surgical complications. In this condition it affects the basement membrane of the capillaries and cell permeability; interfering with the transfer of oxygen and nutrients to the tissues. Along with decreased supply of leukocytes into this area and also high concentration of glucose in the tissue fluids which help in the growth of pathogenic organisms, it impairs wound healing. The vascular changes that take place during diabetes are responsible for ulceration of the foot. The principle finding in the vascular flow mechanism in diabetes is a marked decrease in blood flow as shown by Doppler waveform. There is associated loss of the important diastolic back

flow phase(high ankle / brachial Doppler ratio) found in diabetes. The major cause of this blood flow change is the distal AV shunting and sympathetic dysfunction. The other cause being atherosclerotic stiffening of the vessels, sometimes this is associated with calcification of the vessel walls. The irregular pattern of blood flow definitely has an influence in the ultimate delivery of oxygen to the tissues.

Diabetic patients also tend to develop calcifications more commonly and at a much younger age. This causes some vascular narrowing which may cause distal ischemia. Calcification of intima may produce trauma to blood cells and cause development of thrombi which may distally produce localized areas of ischemia and gangrene.

Histologically the disease is manifested as a thickening of the tunica intima along with large atheromatous plaques and lumps. The metatarsal arteries are more often occluded in diabetes.

An important yet a very common factors responsible for producing neuropathic ulceration is the presence of diffuse distal peripheral polyneuropathy due to which painless ulcers are predominantly seen in the lower limbs. During this type of ulceration there is involvement of all types of nerve fibres. A notable feature is the marked reduction in the amount of Schawann cells. Neuropathy is predominantly selective for large myelinated fibers sub serving proprioception, touch and vibration senses. Later small

myelinated fibers and non-myelinated fibers sub serving pain and temperature sense are affected leading to mixed fiber neuropathy. Osmotic and metabolic derangements are caused due to ischemia from vessel occlusion and altered capillary permeability; this paves the way for producing neuropathy. Increased capillary permeability allows the toxic proteins which are circulating to reach the nerves and tissue oedema which cause the impairment of nutrition contribution to development of the neuropathy.

Metabolic conditions prevalent are more important in the development of the neuropathy than are the vascular diseases. Abnormal Schawann cell function causes segmental demyelination, which is a dominant histological aspect of diabetic neuropathy. This hinders nerve conduction and the situation can only be reversed by remyelination. There are number of reasons which contribute to the production of myelin. Some of these factors are:

1) Synthesis of abnormal myelin
2) Increased activity of polyol pathway in conversion of glucose to fructose with a resultant increase in concentration of metabolites. Simultaneous occurrence of axonal damage is also observed, but segmental demyelination of diabetic neuropathy may exist independent of axonal damage. Axonal damage is the first manifestation of diabetic neuropathy. Normal nerves contain high concentration of myoinositol

and a disturbance in its metabolism in diabetics is suggested as the mechanism for axonal damage.

WAGNER'S CLASSIFICATION

GRADE 0:NO ULCERATION(BONY DEFORMITY,HIGH RISK GROUP).

GRADE 1 : SUPERFICIAL ULCERATION.

GRADE 2 : DEEP ULCERATION WITH PENETRATION INTO TENDONS, BONES, JOINTS.

GRADE 3 : DEEP ABSCESS / OSTEOMYELITIS.

GRADE 4 : GANGRENE OF TOE / TOES / FOREFOOT.

GRADE 5 : EXTENSIVE GANGRENE FOOT / LEG.

The absence of the sweat glands or its malfunction, with the loss of Lysozymes alters the cutaneous bacterial environment and also decreases the defense mechanism of the skin. Trophic changes include redness and glossy shining skin with loss of hair. Thick keratinization of the skin and atrophy of the local subcutaneous tissue is observed in areas of increased pressure. Breakdown of such skin produces trophic ulcers.

Tropic Ulcers

Tropic ulcers are particularly perforating ulcers of the foot, which is associated with Tabes dorsalis, diabetic neuropathy, leprosy, and other diseases of the central nervous system.

About 10 to 20% of the patients with leprosy all over the world complain of the most common yet the most serious complication that is of ulceration on the bottom of their feet.

The second type of ulcer which is seen here is the stasis ulcer which is seen in the crease of flexor muscles of the ankle, due to autonomic dysfunction leading to abnormality in regulation of blood supply and absence of sweating. The last and the third type of ulcer seen in leprosy is the plantar ulcer, which is seen in the weight bearing areas of the foot where there is sensory loss (Dharmendra).**19**

The three modalities of neurological deficit are:

1) Sensory loss.
2) Motor paralysis.
3) Autonomic nerve damage.

Sensory loss

Impending danger to the tissues is preceded by the sensation of pain, which is the body's natural alarm system. But in the decreased condition due to the neurological impairment (sensory loss), repeated trauma may attain proportions which might actually cause tissue destruction.

Motor Paralysis

Only open injuries on the foot with intrinsic paralysis are usually liable to be ulcerated, whereas the foot prone to ulcers always shows motor as

well as sensory loss. The pads of the fore foot are protected by the intrinsic muscles of the foot in two ways, one by straightening the toes, the metatarsal heads are slightly retracted and the pulps of the terminal segments also take a small share of the weight. When these muscles are atrophied the bony prominences are made more obvious.

Autonomic nerve damage

a) Blood supply

The regulation of the blood supply to the sole of the foot is largely, reflex, depending on the integrity of sympathetic supply to the muscular part of the arteriole. In the nerve trunk lesions, these nerves are also destroyed so that the arterioles are sympathectomized. In an anesthetic foot, the increase in blood supply after exercise or slightly excessive stress is not present and hence there is a relative tissue anoxia ensues.

b) Sweat loss

The second autonomic function that is disrupted is the control of the sweat glands.Thus the denervated foot is dry. Due to this deprivation in the supply of the sweat, another one of the skins protective mechanisms is lost, which causes the skin to loose its suppleness. Deprivation of sweat causes the skin to go dry and crack, which may cause the introduction of infections into the body. This kind of skin cracking can be viewed most commonly in the

weight bearing areas where special mechanical strains are supplied during normal walking.

Studies have shown that the initiation of the ulceration is usually due to an initial damage in the deep tissue surface. The pressure of the entire body falls on the foot and during walking, unperceived by sensation and the normal rotation of the joints is uncontrolled by muscular coordination, the result being the introduction of torsional stress, which leads to necrosis of the deep tissues immediately adjacent to the bone. The clinical features follow a sequence of incidents listed below:

1) Initial deep necrosis.
2) Necrotic sinuses.
3) Necrotic blisters and finally.
4) Plantar ulcer.

MALIGNANT ULCERS

Squamous cell carcinoma

This is the carcinoma of the cells of the epidermis that usually migrate outwards to the surface. The initiation point of the squamous cell carcinoma is usually the layer that is formed by the prickle cell layer. This may also occur in a few preexisting lesion of the skin like:

1. Long-standing chronic ulcers (e.g. Marjolin's ulcers) following burns, venous ulcers, old scars etc.

2. Senile keratosis.
3. Bowen’s disease.
4. Leukoplakia.
5. From skin exposed to irradiation.
6. Chronic skin lesion e.g. Lupus Vulgaris (Cutaneous tuberculosis), eczema, warts.
7. Exposed to prolonged irritation by various chemicals.

Very rarely squamous cell carcinoma may develop from a basal cell carcinoma.

Two types are usually seen:

1) Proliferative type.
2) Ulcerative type.

The ulcerative type is found to more commonly prevalent.

Macroscopic Features

The origin of the squamous cell carcinoma is as a small lump or nodule. These nodules enlarge as they mature and become necrotic and their centers sloughs out. The shape of the ulcers is usually circular or oval, and their shape and sizes varies extremely. The edges of the ulcer are raised and everted; this shows excessive tissue growth just above the surface. The floor of the wound is covered by necrotic tumor, serum and blood. Presence of pale coloured unhealthy granulation tissue may also be seen. Sometimes deeper

structures such as muscle, tendon, cartilage or bone may also be seen. The base of the wound is usually indurated.

Microscopic Features

Solid columns of epithelial cells, which are seen, growing down into the dermis, separated from one another by connective tissue. These extend into bulk like masses, which in section appear to be distinct and detached. In the course of time in which the wound evolved the cells lying nearest to the center of the wound, being the oldest undergoes degenerative changes and get converted into hyaline structure less masses of keratin. This process is called keratinization. The mass of keratin looks red with eosin stains. This is surrounded by normal looking squamous cells presenting the characteristic 'prickle cell' appearance and these are arranged in collective manner as seen in 'onion skin'. This whole arrangement is called as a 'cell nest' or 'epithelial pearl'.

The appearance of the cell nest is a characteristic feature of epidermoid carcinoma, but this may be absent in rapidly growing tumor and in mucous membranes.

Another important feature to be noted in this type of malignant tumor is the infiltration of the dermis by chronic inflammatory cell particularly plasma cells.

Malignant Melanoma

The originating point of the malignant melanoma is a malignant lesion in the melanoblasts of the skin. All the melanomas originate from the melanoblasts at the dermo-epidermal junction, but as the cells may not contain melanin at all times and therefore some lesion may be amelanotic.

The malignant melanoma may arise from a pre-existing pigmented naevus (90%) – either a junctional naevus, compound naevus or in Hutchinson's Lentigo.

Macroscopic Features

The epithelium lying just above the mole ulcerates and often breaks down with minor trauma, which makes the wound to bleed. Palms and the soles are the most common areas where melanomata are seen. The coloration may range from black to brown. If the wound is more blackish in colour the chances of it being malignant is more. When they are small, the surface is smooth but as it attains larges size, ischemic necrosis occurs and small ulcers and crusts etc, are seen on the surface. The surface of the big melanoma appears wet, soft and boggy yet the tumour feels firm.

Microscopic Features

Development of the malignant melanoma brings forth an increase in the functional activity and the cells increase in size. The nucleolus is enlarged, hyperchromatism is present and mitosis is observed. The proportion of

nucleus to cytoplasm is also increased. The cytoplasm is often vacuolated with fine melanin granules to mimic paget cell. These changes can be seen through the epidermis. To determine whether the naevus is malignant or not the surface layers in the epidermis are examined for presence of cells which are vacuolated. The invasion of the dermis is determined by the presence of polyhedral or circular cells with abundant spongiocytoplasm and fine pigment granules. The tumor cells may form clusters in the sub epidermal lymphatics a dreadful indication of early stage of lymphatic spread. Invasion of lymphocytes which are inflamed are found in the sub epidermal zone. This is a very good indicator of a malignant melanoma. In fbully developed melanoma the large tumor cells in the dermis often show alveolar arrangement the groups being separated by a fine stroma.

VII. DIAGNOSIS

Chronic Foot ulcers can be diagnosed based on the patient's medical history, appearance and location of the ulcer along with clinical examination and investigations.History is very useful in diagnosing ulcers caused by associated diseases like Diabetes Mellitus, Syphilis, Tuberculosis,Renal disease, Liver disorder.

The primary lesion might have been altered either by the use of ointments or by infections. Thus a history of eczema helps for diagnosis of stasis ulcers. History of trauma will help for evidence of underlying fractures

or osteomyelitis. A nodular ulcer appearing after a thorn prick points to deep mycosis. A painful ulcer with undermined edge or ulcer healing at one end and growing at other end might be of tubercular origin. Ulcers growing on already existing moles may be malignant melanoma. Rolled out borders of chronic ulcers may be carcinomatous ulcer. Ulcers growing on scar tissue may be Marjolin's ulcers. Punched out ulcers without any symptoms in elderly people may be due to gumma. Knowledge of precipitating factors is also equally important. Ulcers that get aggravated during cold season indicate Raynaud's disease. Thus a clear idea of primary lesion of an ulcer and its later development helps one in diagnosis.

An understanding about the location of the ulcer is crucial. The area around the medical malleolus is drained by long saphenous venous system and communicating vein of lower limb and this is the area where occurrence of stasis ulcer is invariably seen. Hypertensive ulcers or ischaemic ulcers are usually pale with an eschar and are usually seen on the lateral side. Hyperpigmentation and or eczematization point towards stasis element. In Werner's disease or scleroderma, sclerotic skin formation is observed.

Atrophic skin formation around an ulcer indicates to acrodermatitis chronic atrophicans or an Atrophic Blanche. Occurrence of the gummatous ulcers is over the sub-cutaneous bones such as tibia, sternum etc.

Distinguishing between a spreading ulcer and a healing ulcer is extremely important. Inflamed and oedematous edges of the ulcers indicate spreading ulcers. In a healing ulcer, the edge is traced from red gramulation tissue and at the center it will show a blue and a white zone. An undermined edge is usually observed in a tuberculous ulcer and punched out edge in gummatous ulcer. A pearly white beaded edge which is raised is characteristic of a rodent ulcer. An everted edge points to squamous cell carcinoma.

An understanding of the floor of the ulcer or exposed part of the ulcer is extremely important. Red granulation tissue covering the floor of the ulcer points to a healing ulcer. Smooth granulation with a pale coloration indicates to a slow healing ulcer. Trophic ulcer penetrates down to the bone which becomes the floor in this case. Black mass on the floor suggests malignant melanoma.

Discharge

The quantity and the coloration of the discharge from an ulcer help in identifying the nature of the ulcer. Profuse discharge from an ulcer indicates to a healing ulcer whereas a purulent discharge points to a spreading ulcer. A greenish coloration to the discharge indicates to an infected ulcer and this may be caused by pyogenic bacteria. Serosanguinous or blood mixed discharge is pathognomonic of malignant ulcer, or else tuberculous ulcer.

VIII. CLINICAL FEATURES AND DIFFERENTIAL DIAGNOSIS

Arterial ulcers are rare compared to the venous ulcers. In arterial ulcer skin is often dry and scaly or shiny and atrophic with brittle nails or loss of hair growth. The appearance of the ischemic erythema is usually on the foot. Atherosclerotic arterial ulcers are often seen in older people and when they occur in the young adults, it usually points to Thromboangiitis Obliterans and other forms of vasculitis. Pressurized areas of the foot are the focal points for the appearance of these ulcers. Toes, bony prominences or in pretibial and lateral malleolar areas are some of the common placer where these ulcers can be spotted. These ulcers have sharp defined edges and are punched out. The ulcer destroys the deep fascia and may even expose the tendons at the base. The ischemic ulcers are extremely painful and the pain is excruciating irrespective of the size of the ulcer. The development of the arterial ulcers is more prolonged when compared to the venous ulcers. The most common forms of trophic ulcers seen are the bedsores. The heel and the ball of the foot and the back of the heel are the most common places where these types of ulcers occur. These ulcers start with callosity under which suppuration takes place, the pus comes out and the hole at the center forms the ulcer which gradually burrows through the muscles and the tendons to the bone. Tuberculous ulcer develop when cold abscess from bone and joint tuberculosis break out of the surface. Appearance of the ulcer is in the form of

a thin edged one wound, which is reddish blue in colour and undermined. The base of the wound is coloured pale and there is slight induration at the base.

The areas where gummatous ulcer appear most commonly is in the upper part of the leg i.e., over the subcutaneous bones of the tibia. The wound has punched out edges and a yellowish grey gummatous tissue (wash lather slough) on the floor.

. The skin of the ankle tends to become abnormally sensitive to temperature in such patients. When the temperature drops the ankle becomes cold and blue and often tenderizes. In hot weather chronic reactive hyperemia is evident and the ankle becomes hot, oedematous, swollen and painful. The patient is much more troubled by chilblains. Palpation of the leg reveals the presence of small, superficial and painful nodules which are numerous. These nodules break down to form ulcers.

The hypertensive ulcers or the Martorell's ulcers are a condition which appear in older aged people and is usually associated with atherosclerosis. This ulcer is extremely painful and is usually punched out and extends down into the deep fascia. It is note worthy that all peripheral foot pulses are present.

The clinical characteristic feature of the Meleney's ulcer is the burrowing nature, which is the undermining of ulcer with a lot of granulation

tissue on the floor. These ulcers are painful and show a tendency to spread and render the patient toxemic.

Ulcers secondary to Paget's disease are mostly situated over the convexity of the anteriorly bowed tibia, with the edges densely adherent to the bone which forms its base.

In case of rheumatoid arthritis the ulcer forms due to break down of the nodules. The size of the ulcer varies, the wound becomes punched out, shallow and is usually painful, without induration and slow to heal.

Ulcers caused by mycotic infections are rare. Primary neoplasms of the skin which may cause ulceration in the lower extremity are squamous cell carcinoma and malignant melanoma.

The occurrence of the squamous cell carcinoma is most common in the age group of 40 years and above, the risk of contracting it raises as the age increases. In this type of ulceration bleeding is a common complaint. The tumor is painless but becomes painful if it invades deeper structures. The origin of the squamous cell carcinoma is as a tender nodule, which enlarges and the center becomes necrotic and sloughs out. The variation of the size of the ulcer is diverse and the wound is everted and has raised edges. Pale and unhealthy granulation tissue may also be seen. The base of the ulcer is indurated, with restricted mobility. Once the tumor is anchored to the underlying structures the regional lymph nodes may become enlarged. But

until it is proved otherwise, it should be assumed that the palpable lymphnodes are due to metastasis.

As explained previously in the malignant melanoma, the patient usually complains of a long-standing mole which has shown rapid growth within a few days. The mole becomes darker and such color change is often patchy. Sometimes malignant melanoma does not show pigmentation and such lesion is called amelanotic ulcer. The overlying epithelium becomes ulcerated and often breaks down. The tumor hence tends to bleed. The tumor cells gradually tend to invade the surrounding skin and thus forming a halo. This condition is not a painful one, but it is often itchy. Only in the later stages does one complain of weight loss, dyspnoea or jaundice. The occurrence of these melanomas is usually in the palms or the soles of the feet. Apart from the bleeding, the surface of the wound looks wet, soft and boggy. The tumor is firm and can be easily lifted from the deeper structures. The regional lymphnodes are often enlarged in malignant melanoma.

In patients suffering from non-specific yet chronic ulcers, the patients usually have a history of trauma which he might have neglected or for which he had taken inadequate treatment. The patients in such cases are of poor build, poorly nourished and anemic on examination. These ulcers appear with irregular margins surrounded by oedema and there may be slight tenderness.

Often ulcers are foul smelling and with seropurulent discharge. Associated induration, sclerosis and pigmentation are common.

IX. MANAGEMENT

a. INVESTIGATIONS

There have been many investigations done for analysis and understanding of the leg ulcers. These investigations are numerous and varied. These investigations help us to come to a definite diagnosis of the ulcers and may also guide about the outcome of surgery if any.

The investigations can be broadly classified in to three different classes:

a) **Routine investigation**

The routine investigations themselves are comprised of the following tests:

- Hemoglobin (Hb%): This test helps to determine whether the patient is anemic where healing is impaired or poor
- **ESR**: ESR is increased in many conditions of various types, but ESR more than 30mm/hr makes one to think about chronic granulomatous disease such as tuberculosis, sarcodosis deep mycosis, SLE, dermatomyositis etc. But ESR has only prognostic value.
- **TC and DC**: increased total count always denotes the increased activity of the reticuloendothelial system. It represents acute or chronic infections. In some cases it can be of vital importance as in Leukaemia. But by itself TC is not sufficient for diagnosis. DC is also taken along

with TC, Differential count tells about the type of cells in increase and also the presence of abnormal and malignant cells. Increase in percentage of lymphocytes indicates chronic infections or allergic phenomenon.

- **Haemogram**: any abnormal findings from the above investigations may call for a complete haemogram to know the further details of the blood cells, e.g. Sickle cell anemia.

b) **Special investigation**

Many different types of special techniques are also used which are classified in the special investigation group.

- **Fasting blood sugar, PPBS, GTT and HbA1c**

These tests are done to determine the presence of diabetes mellitus.

As we know diabetes mellitus is one of the many contributing factors for the formation of ulcers in legs and for peripheral neuropathy. The ulceration process is hastened in the presence of higher sugar concentration in the blood.

- **Bone Marrow**

Bone marrow aspiration is recommended in cases where leukaemia or aplastic anemia is suspected. The use of the sternal bone for this test is usual practice. Presence of abnormal blood cells and an increase in the WBC content and their precursors help in the process of diagnosis.

- **Lipid profile**

The detection of the atherosclerotic condition is possible when this particular test is performed. It has been noted that people without arterial disease maintain a LDL cholesterol level of 130 mg/dl or less. A LDL level of more than 160mg/dL must be treated with lipid lowering drugs.

- **Lupus Erythematous cell test**

The presence of systemic lupus erythematosis is confirmed by performing this particular test. In recent times this test has been replaced by other tests such as anti DNA antibodies test and L.E band test. The test is very helpful in detection of lupus Erythematosis and other tissue disorder.

- **Blood VDRL test**

The VDRL test is a non-specific flocculation test used for diagnosis of syphilis, Yaws Pinta and Bejel. The test depends on the presence of Reagin antigen in blood and it cannot differentiate Yaws or Pinta from syphilis.

- **Liver function test**

The status of liver and its functioning is known by the performing this test.

- **Culture and sensitivity of Bacteria/fungus**

The pus from the ulcer is sent to the lab for culture and sensitivity for bacteria or fungus as the case may be.

The presence of these pathogens in the pus does not have to mean that they are the causators. These pathogens only act as catalysts and worsen the

condition and hinder essential processes such as coagulation and fibrinolytic activity from taking place. Presence of some bacteria must be taken more seriously. These bacteria are:

a) Heamolytic Steptococci.

b) Fusiform bacilli and spirochete in hot humid conditions.

c) Candida albicans.

d) Pseudomonas aeroginosa.

e) Proteus

f) Staphylococcus

Presence of these organisms needs attention. They have to be treated with proper antibiotics according to culture and sensitivity.

- **Biopsy and histopathological examination from ulcer site**

Malignant skin changes are common in chronic ulcers. A biopsy should be taken from all suspicious ulcers or ulcers that do not respond to appropriate treatment. Also,chronic ulcers need to be biopsied at regular intervals as malignant change in these ulcers in directly related to their duration (Yang D. Morrison B. D. et al and Smith J, Mello I. F et al).**20,21**

- **Radiological examination**

X-ray should be taken to find out any bony abnormalities and or osteomyelitis of the underlying bone.

c) **Investigation of vascular structure of the leg**

Doppler ultrasound

A Doppler flow probe can be used to exclude arterial disease and to determine patency of a vein and a bi-directional probe is used to detect any reflux. This investigation is carried out with patient standing. Doppler probe is first placed on sapheno femoral junction (SFJ) and blood flow is assessed to located venous flow in common femoral veins. With one hand the examiner gently heard as “woos” from loud speaker of the Doppler machine. The calf compression is released and any backflow is noted. The probe may be also held in sapheno-popliteal junction (SPJ) while calf is compressed and released to test competence of veins in this region.also useful in identifying arterial thrombus and stenosis

Duplex ultrasound imaging

This technique involves use of high-resolution B-mode ultrasound imaging and Doppler ultrasound to obtain images of veins and simultaneously measure the flow. It Provides both functional and anatomical information. Modern duplex machines represent Flow as a colour map which is superimposed on grey scale image of the vessel. This technique is highly reliable in the investigation of arteries and veins, and is most appropriate when detailed analysis of anatomy and physiology of venous system is required. Blocked or incompetent veins can be easily identified by biphasic flow. Reversal flow, in venous reflux can be recognizable by a change in

colour, so that incompetent veins and leading walls show up as ‘red’ at the moment of reflux.

The origin of varicose veins and venous ulceration can be identified and in patients with deep vein thrombosis the thrombus can be seen (Seurr John. H et al).**22**

Plethysmography

a) Photo- Plethysmography: a probe is attached to the skin to asses venous refilling of surface venules by measuring light transmission of the skin. The filling of vessels reflects pressure in the superficial veins of the leg. The patient sits till the trace stabilizes. Then he performs a series of 10 dorsiflexion at the ankle. The venous pressure falls as venules empty and the trace falls. The patient sits and venous refilling occurs only through arterial inflow, a slow process taking 20 to 30 seconds when the limb is at rest. In venous incompetence filling occurs through venous reflux also which speeds the filling time.

b) Other forms of Plethysmography like air plethysmography, and strain gauge plethysmography are used only by experts in laboratories to quantify venous function caused by incompetent veins.(Seur John. H et al).**22**

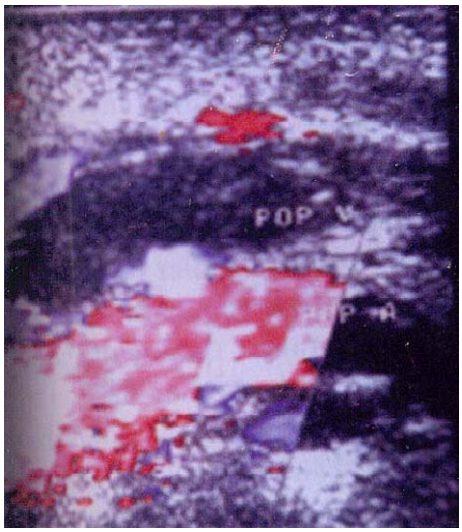


Fig 8: Thrombus in popliteal vein

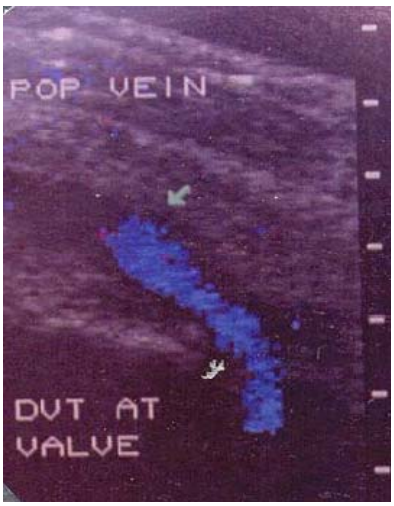
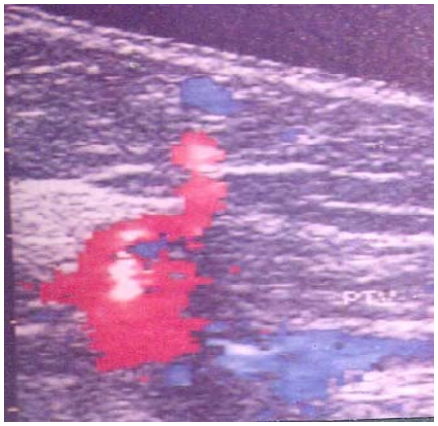


Fig 9: Popliteal vein with thrombus near the valve



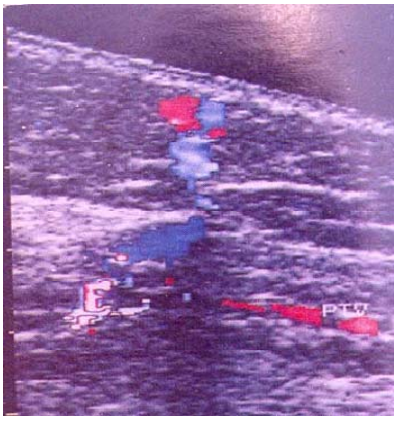


Fig 10: With distal compression of foot, flow is normal from superficial to deep vein (coded red), on release flow is retrograde through the perforator (coded blue)

Fig 11: Superficial artery occlusion

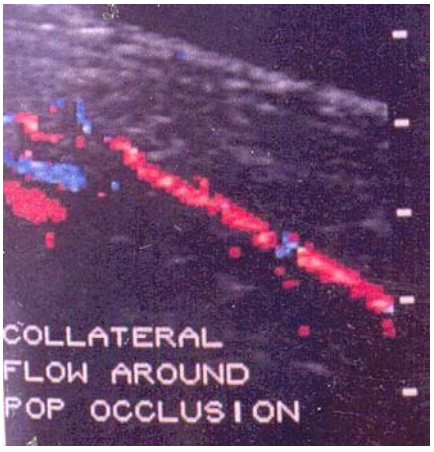


Fig 12: Popliteal artery with collateral flow around

Radioisotope tests

Fibrinogen labelled with radioactive isotope I^{125} is injected into the patient. This is converted into fibrin by thrombin. Fibrin is deposited in thrombus.

Various other tests are also performed in this type of special investigations. Some of these are mentioned below:

Arteriography

Arteriography is indicated only when decision has been taken that intervention is needed. Arteriography involves injection of radio opaque solution into the arterial tree using retrograde percutaneous method involving femoral (Seldinger technique). Direct angiography by puncturing the aorta is hazardous which include the thrombosis, arterial dissection, haematoma and anaphylaxis, hence outdated (Murie. John).**24**

Digital Subtraction Angiography (DSA)

This technique involves a computer system to digitize angiographic information. It allows image before contrast injection to be subtracted from contrast image, yielding great clarity. This avoids arterial puncture although high volumes of contrast agents are required.

b. TREATMENT

The main aim of treating any ulcer is to make it heal and to treat its underlying conditions. Various methods of treatment are prevalent today. The

treatment given to the patient can be classified into three general classes. They are explained in detail below.

General Treatment

In treating the ulcer the surgeon must first recognize and treat any systemic condition that inhibits the healing. Systemic diseases, particularly malignant tumors, diabetes mellitus, nutritional deficiencies and some medication can retard the wound healing. In addition deficiencies of vitamin C, zinc, iron and protein can also retard healing as these are the nutrients needed to synthesize collagen. It is important to keep in mind that some medications like systemic glucocorticosteroids; cytotoxic drugs and nonsteroidal anti-inflammatory drugs if administered may cause impairment to the process of collagen production which intern hampers the wound healing process.

The general practice of treatment of the patient with co-morbid conditions must include rising of the patients limb to an elevated position and performance of exercises within bandage supports. The elevation of the affected limb well above the heart level is an excellent treatment method and the limb must be allowed to remain in this state for at least 2 hours. The surgeon must encourage the performance of exercise. The exercises need not be too vigorous and might just be simple ones as rotation of the ankle through full range and gentle lift of the leg off the floor. The rate of exercise should be

around 40/min and this is enough to empty the veins without overloading the lymphatic system.(Bisgaard's regimen)

As a rule elevation must not be done in cases of ulcers of arterial origin.

Systemic treatment

Infection of the ulcer wound greatly hampers the wound healing process and keen attention must be paid when dealing with such cases. The treatment of such ulcers must employ the use of proper medication in the form of antibiotics. Ideally antibiotic therapy according to pus culture and sensitivity is appropriate.

Oral Zinc Sulphate Therapy

The value of this method in the process of ulcer treatment remains debatable. But it is reasonable to treat patients who have shown to have low plasma levels or where there is a background of malnutrition or an inadequately balanced diet, prolonged anaemia or toxemia. A dose of 220 mg twice a day is well tolerated.

Vasoactive medication

Some vasoactive agents have been found to be effective in various conditions. Nifedipine is effective in Raynaud's disease as a result of its ability to restore blood flow to the digits. Ketanserine, an S-serotoninergic blocker is helpful in treating ischaemic ulcers. Pentoxifyline (trental) is effective in treatment of the above conditions. Other physical modalities of

treatments such as transcutaneous electrical nerve simulation may also act by increasing blood flow.

Ketanserine has also been shown to increase the rate of ulcer healing in diabetic ulcers (O'Meara S.O. Cullum N et al).**25**

Local treatment

Local application

This method is practiced to soak up exudates, remove slough, control infection, sooth the surrounding skin, promote granulation tissue and protect seeding of new epithelium as well as to relieve pain. The effect of this type of treatment remains marginal or insignificant if other methods such as elevation, exercises are not administered.

It is not always possible to heal an ulcer completely, so the next most logical thing must be done that is it should not be allowed to get worse. To accomplish this relatively bland agents such are used on the ulcers to control the incurrence of infection. The use of topical antibiotics must be restricted as most of these are strong sensitizers. The use of antiseptic agents is safer and acceptable.

Wet dressing using Eusol or 0.5% Acetic acid, chlorhexidine or 0.5% silver nitrate have been shown to be inhibitory to granulation tissue, and must be advised with caution. However, Eusol is a useful wetting agent for softening and removing superficial slough, especially when there is no

granulation tissue. 0.5% silver nitrate is recommended when the granulation tissue is over exuberant. Cautious irrigation with saline or repeated application for the removal of saline soaked gauge may be helpful.

Gilliland E. L and Wolfe John H.N.**7** recommended povidone as the antiseptic for general use and silver sulphadizine for short periods of pseudomonas species have been cultured and potassium permanganate (1/8000 dilution) for wet ulcers with surrounding eczema but again only for limited periods as it can cause local hyperkeratosis.

In recent time's new topical dressing have gained wide acceptance in the treatment of foot ulcers. These products can optimize wound healing by providing moist environment, which aids keratinocyte migration over the surface of a wound. The hydrocolloid dressings are to debride the ulcer, while some of the polyethylene dressings have the advantage that they decrease the pain in the ulcers. These dressings do not increase the risk of infection in the ulcer colonized with bacteria.

Carboxy-methyl-cellulose dressings are safe, effective and well tolerated in management of non-ischaemic, non-infected deep diabetic foot ulcers (Piaggesi A., Baccetti F et al).**26**

Compression bandages

Bandages are either used to compress or to retain dressings, support and contain any prior reduction and swelling. The advantage of compression

lies in the provision of adequate counter pressure when the patient continues normal occupation and normal life. Full use of natural leg pump is encouraged.

Compression is more effective in healing venous leg ulcer than is no compression and multilayered high compression is more effective than single layer compression. High compression hosiery is more effective than moderate compression in preventing ulcer recurrences (Callam N, Nelson E. A et al).**27**

SURGICAL TREATMENT

Many ulcers that are infected and do not heal require wound toileting debridement and slough excision. Those ulcers which still do not heal by secondary intension or have delayed healing or those which have large areas of defects require skin grafting.

Grafts

Primary indication for split skin grafting is the failure of conservative line of management. It is also helpful in cases of extensive ulcers which when treated conservatively will take long drawn-out period for healing; that too with fibrosis and scarring.

Split thickness grafts

These can be used in larger ulcers. It is advisable to use a meshed graft that allows exudates to escape. This technique requires extensive local or spinal anesthesia. The donor site may be painful and slow to heal. Meshed

graft heals by secondary intension, which results in irregular surface of the grafted site.

Keratinocyte grafts

Cultured epithelial grafts were first shown invitro by Ronwall and Green and first used clinically by Gallico in 1984. Here only epithelial component can be cultured and dermal component is lacking. Their lack of dermal component restricts adherence and attachment of underlying tissue reducing long-term durability of the graft. Work is also being done on dermal substitutes that can be used as temporary dressing. A combination of epidermal, dermal skin substitute holds great promise, at present, for future success (Henry C. Vasconez).**28**

Autografts : Human keratinocytes from a small skin biopsy specimen can be grown in vitro, in 2 to 3 weeks, to form sheets of confluent epithelium.Cultured autografts have also been applied to chronic non-healing ulcers. They seem to provide rapid healing in some cases.

Allografts: Culture of epithelium derived from an allogenic donor (new born fore-skin) and subsequent application to chronic non-healing ulcer are fairly recent developments.

Cultured allografts have been found to promote healing in burns. The new born keratinocytes appear to provide a potent stimulus to heal difficult skin ulcers of varying etiology. Cryopresentation of these allografts allow

storage of readily available grafts in skin banks. Cultured allografts may prove to be useful alternative for this treatment of chronic ulcers.

In chronic wound such as ulcers, however both auto grafts and allografts seem to act as temporary biologically active wound dressings. It is likely that growth factors released by the keratinocytes stimulate healing.

Also cultured epidermal allografts hasten healing of refractory leg ulcers apparently by stimulating the migration and / or proliferation of acceptors keratinocytes rather than through take up of the graft (Beele H., Naeyaert J. M. et al).**29**

SPECIFIC TREATMENT

Venous ulcers

Specific treatment of venous stasis diseases must first be directed towards reducing venous hypertension. Patients with venous stasis diseases should wear support hose, but for patients who cannot wear support hose, treatment with Unna boot has shown to help healing venous stasis ulcers. These boots consist of zinc oxide, calamine and glycerin.

The hypertension can also be reduced through the use of intermittent pneumatic compression.

Once the ulcer has healed, to prevent recurrences specific surgical procedures in form of saphenofemoral ligation (Trendelenburg's operation)

subfascial ligation, incompetent valve reconstruction will become necessary. A bypass operation may be performed for thrombosis.

Arterial ulcers

The therapy must be individualized. If the ulcers result from the localized occlusion of small arterial branches, then some will heal solely with bed rest with elevation of the head end of the bed and cool saline compression.

Others will respond to lumbar sympathectomy(which is for palliation and not curative) combined with complete excision of the ulcers and its surrounding rim of infarcted tissue followed by a split thickness skin graft.

Occasionally if arterial bypass surgery may be warranted, it is of utmost important to determine the arterial inflow at the iliac level is adequate before attempting reconstructive arterial surgery on the femoropopliteal system.

Transluminal angioplasty may be performed for occlusive disease by inserting a balloon catheter into an artery and inflating it within a narrowed area. To a lesser extent the vessels of the leg itself may be dilated with a good outcome followed by stents. Artherectomy can be performed by a variety of new devices available to allow the percutaneous removal of atheroma from within the vessel.

Diabetic ulcers

Proper assessment of the diabetic status, severity of infection and general nourishment is the key to the success of management of ulcers.

Regardless of the type of diabetes mellitus, they are suffering from (IDDM or NIDDM) all patients must be put on soluble insulin therapy. Depending upon the blood and urine sugar measurement doses should be adjusted.Control of infection is achieved by using combination of broad-spectrum antibiotic agents depending on the pus culture and sensitivity.

Malignant ulcers

- ❖ Squamous cell carcinoma, malignant melanoma.
- ❖ Management of squamous cell carcinoma can be considered under the following headings.

a) Treatment of primary lesion

b) Treatment of secondary lymph nodes

a) Treatment of primary lesion

Wide local excision is the treatment of choice once the diagnosis is confirmed by biopsy. Excision of the growth should be performed with 2 cms of the normal tissue surrounding the tumor in width and depth.

In case of tumor is involving the toes amputation or disarticulation is indicated.

Superficial radiotherapy may cure 80% of early lesions. Different forms of radiotherapy may be applied according to the size and type of tumor. Such methods are deep X-ray therapy, radium needles and moulds. Radiotherapy is helpful where the growth is small, has not involved muscles, cartilage or bone.

b) Treatment of secondary lymph nodes

When there are no enlarged regional lymph nodes – regular follow-up is advised. When the lymph nodes are significant clinically as well as pathologically radical block dissection is justified.

The defect or the gap of wide excision is covered either by split skin graft taking skin from the contralateral limb, as skin grafting from the ipsilateral side may result in recurrence or with myocutaneous flaps and microvascular free tissue transfers.

Treatment of malignant melanoma is primarily surgical. There should be complete surgical excision. Minimum margin of 1 cm and maximum margin of 2 cm should be made. This represents clinical margins at surgery and not histopathological margins. Some surgeons recommend 5cm margin.

Treatment of lymph nodes

Clinically involved nodes require therapeutic node dissection. This should be done by those with expertise in this field.

Lymphatic mapping and sentinel node biopsy is a technique popularized by Mortan in 1994 for melanoma. This may provide an approach to identify those who may be appropriate for elective node dissection by detecting presence of micrometastases in the sentinel node which is tracked by using lymphoscintigraphy following an intradermal injection of radio colloid at the primary site. This accurately indicates presence or absence of micrometastases in a nodal field.

Treatment

Initial line of treatment was to improve the general physical conditions of the patients. Anaemic patients were put on iron preparations and some patients needed blood transfusion.

If patients were dehydrated, fluid and electrolyte balance were restored by appropriate fluids like ringer lactate and isolyte solutions and vitamins were infused along with the fluids. Intravenous protein preparations (Amino drip), and intralipid infusions were instituted if required.

TAO patients were advised to quit smoking and were put on peripheral vasodilators like pentoxyphylin and most of the patients had undergone indirect arterial surgery for palliation (Lumbar sympathectomy). Some patients had underwent Ray’s, Gillies, Lisfranc’s, chopart’s amputation.

Patients who were suffering from varicose ulcers were first treated for ulcer with limb elevation, compression bandaging (Bisgaard's line of

treatment). Later the patients were posted for surgery - Trendelenburg's operation (i.e. flush ligation of S.F. junction) and stripping (Meyers stripping); if there is saphenofemoral incompetence or multiple ligation and excision of vein, if perforators are incompetent cockett and dodd surgery. Some of the patients who were not ready for surgery were treated conservatively with further compression bandaging. (Elastocrepe bandage).

Diabetic patients with ulcers present in late stages with their random blood sugar >300 mg/dl and some patients with even more and these were put on diabetic diet which was rich in proteins and vitamins. Urine sugar was kept as near the base line as possible with insulin therapy. Plain insulin (Bovine) was initially used according to sliding scale and later a fixed dose was maintained in consensus with the physician. Local treatment of lesion by cleaning and dressing was done. Slough excision was done, pus and discharge was sent for culture and sensitivity, and treated with antibiotics accordingly. Various local ointments used in the treatment included that of povidone-iodine, collagenase ointment (salutyl) and antibiotic containing ointments (Metrogyl ointment). Neuropathy was treated with neurotropic drugs.

Patients with atherosclerotic occlusive disease and increased lipid profile parameters were treated with lipid lowering drugs in consensus with the physician. They were further referred to vascular surgeon for direct arterial interventions.

Amputation was resorted to in cases of extensive gross infection and tissue necrosis, where it had been done as a life saving procedure. Malignant ulcers were confirmed with edge biopsy and were treated with wide excision. Raw areas were covered with split skin graft.

By the time of discharge some cases with residual wounds were advised to come for dressing twice a week.

Diabetic patients were suggested to attend diabetic clinic regularly after discharge from the hospital.

METHODOLOGY

The material for this study was drawn patients admitted to the Surgical Department, Tirunelveli Medical College Hospital, Tirunelveli from March 2011 to March 2012.

A total number of 100 cases were considered for this study. This group was a diversified one and included patients of both sexes and of all ages from 12 years and above, all religion and economic strata. This study included cases of stasis ulcers, diabetics with leg ulcers, arterial ulcers and others.

A detailed history was collected with particular reference to onset, duration and type of lesion, socioeconomic strata and occupational factors and systemic diseases. Any histories of similar ulcers were also noted.

A thorough systemic and local examination was carried out. The morphological features of ulcers i.e. - number, distribution of ulcer on gaiter area or foot site and associated diseases like varicose veins, eczema or patches were noted. But while presenting only relevant positive and some important negative findings were shown to make the study brief and to avoid unnecessary repetitions.

Fig 13: Venous ulcer

Fig 14: Diabetic ulcer

Fig 15: Arterial ulcer (TAO)

Fig 16 : Malignant ulcer (Squamous cell Ca)

Fig 17: Tropic ulcer

ANALYSIS AND OBSERVATIONS

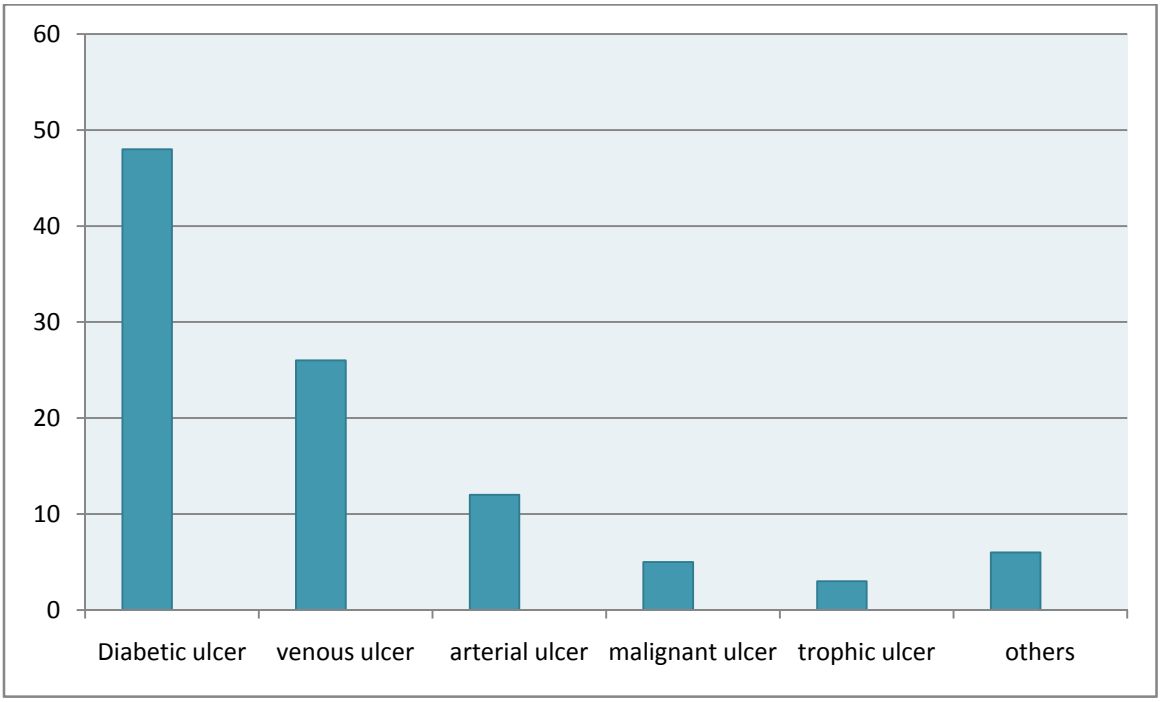
Table 1

Distribution of various types of foot Ulcers

Total No. of patients studied: 100

Sl.No	Etiological Type	No. of patients	Percentage
1	Diabetic ulcer	48	48%
2	Venous ulcer	26	26%
3	Arterial ulcer	12	12%
4	Malignant ulcer	5	5%
5	Tropic ulcer	3	3%
6	Other ulcers	6	6%

Graph 1: Showing distribution of various types of foot ulcers



Among the 100 cases studied the commonest was found to be diabetic ulcer accounting for 48 cases (48%) followed by venous ulcer (26%),arterial ulcer (12%) malignant ulcer (5%), tropic ulcer (3%) and others (6%).

According to Gilliland 95% of leg ulcers are due to vascular etiology and venousulcers dominates accounting for up 90% of the cases. Arterial ulcers account for 5 &10% and others are due to neuropathy or a combination of both (Young RJ).**10**

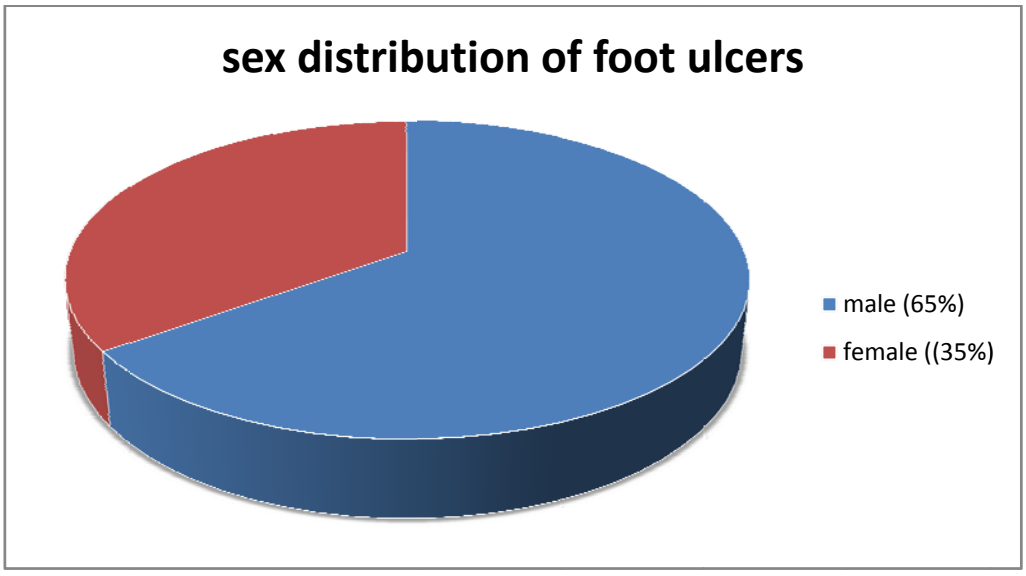
Incidences of various foot ulcers are shown graphically in graph No.1 above.

Table 2

Sex distribution of various types of foot ulcers

SEX	NO. OF CASES	PERCENTAGE
Male	**65**	**65%**
Female	**35**	**35%**

Graph 2: Showing Sex distribution of leg ulcers



The above figures indicate that foot ulcers were more common in males than in females – males accounting for 65%.

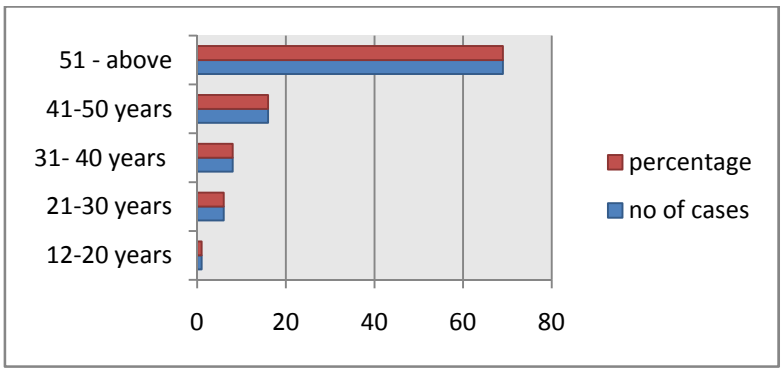
However, in other studies there has been no gross difference between male/female ratio.

Table 3

Age distribution of various types of foot ulcers

Sl. No	Age Group	No. of cases	Percentage
1	12 – 20 years	1	1%
2	21 – 30 years	6	6%
3	31 – 40 years	8	8%
4	41 – 50 years	16	16%
5	51 – above	69	69%

Graph 3: Age distribution of various ulcers



Incidences of foot ulcers in this study group were found to be maximum in the age group of 51 & above. Since, the patients of age group 0 – 12 years are taken care of under the department of pediatric surgery, they are not included in this study.

The youngest patient was 18 years old and the oldest were 84 years old. Cornwall et al**30** in their study had 70% of the patients over the age of 70

years and according to a study done by Callam MJ**27** ulceration began before the age of 40 years in 22% of the patients.

Ulcers associated with diabetes mellitus

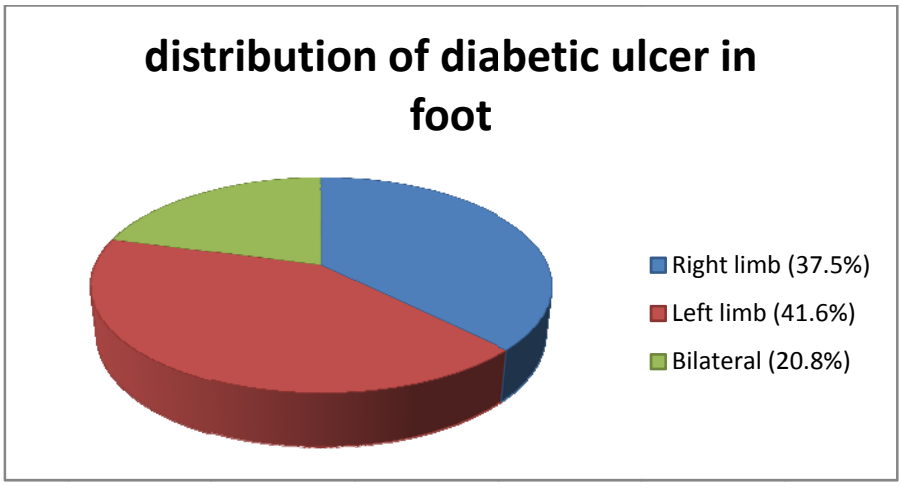
Out of 100 cases studied ulcers associated with diabetes mellitus accounted for 48 cases.

Table 4

Distribution of diabetic ulcers in the foot

Sl. No	Side	No. of cases	Percentage
1	Right limb	18	18%
2	Left limb	20	20%
3	Bilateral	10	10%

Graph 4: Distribution of diabetic ulcers in the foot



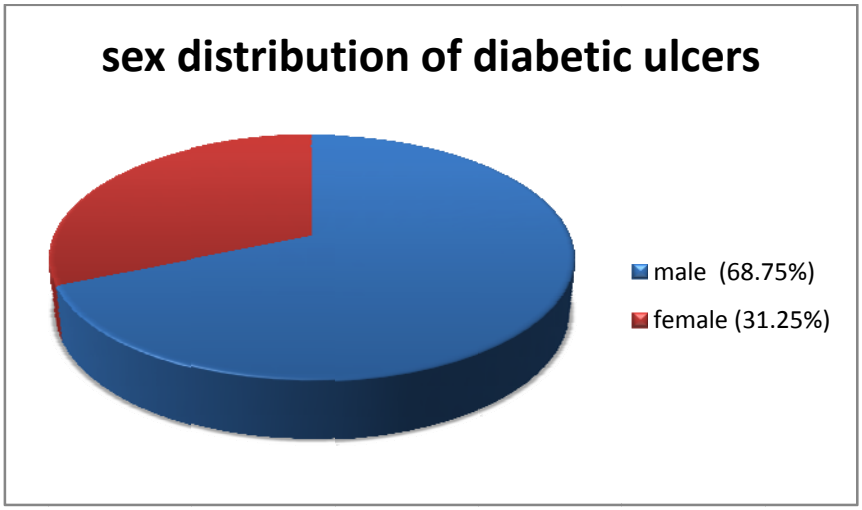
From the above study, it is noted that diabetic ulcers were relatively common in the left limb accounting for 41.6% of cases.

Table 5

Sex distribution of diabetic ulcers

Sex	No of cases	Percentage
Male	**33**	**68.75%**
Female	**15**	**31.25%**

Graph 5: Showing sex distribution of diabetic ulcers



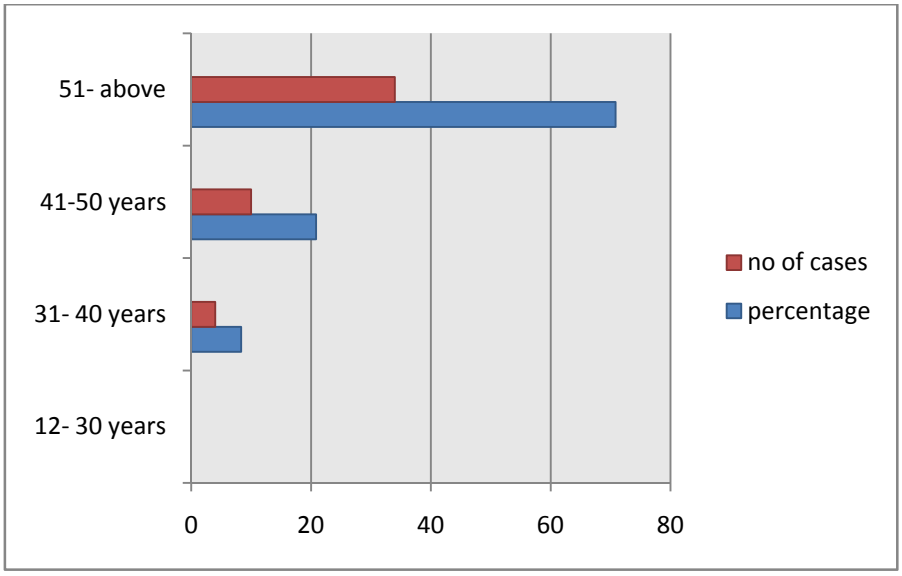
From the above study, it is noted that diabetic ulcers were relatively common in males accounting for 68.75% and less common in females accounting for only 31.25%.

Table 6

Age distribution of diabetic ulcers

Sl. No	Age group	No. of cases	percentage
1	12- 30 years	0	0
2	31- 40 years	4	8.33%
3	41- 50 years	10	20.83%
4	51- above	34	70.84%
	Total	48	100%

Graph 6: Showing age distribution of diabetic ulcer



As noted above the maximum no of patients suffering from diabetic ulcers were in the age group of above 50 years accounting for about 70.84% of the cases.

Venous Ulcers

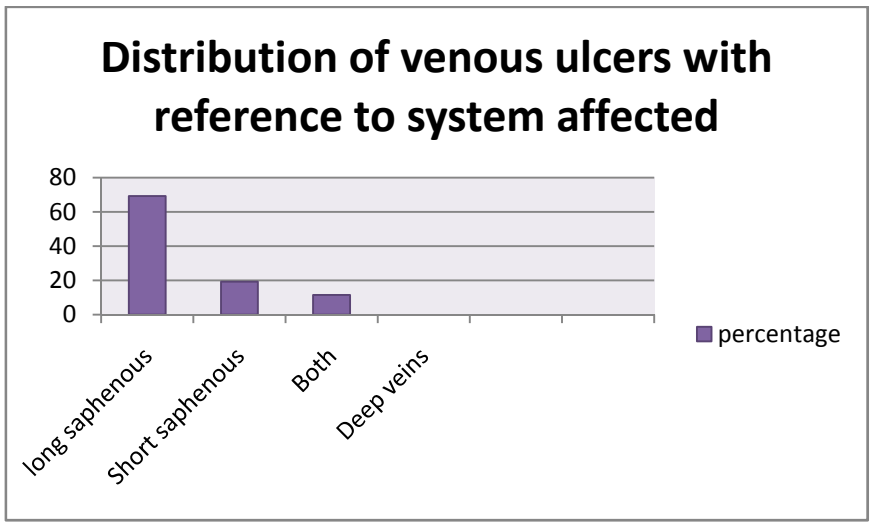
Out of the 100 cases studied ulcers associated with venous causes accounted of 26 cases.

Table 7

System affected in venous foot ulcers

System	No. of cases	Percentage
Long saphenous	**18**	**69.23%**
Short saphenous	**05**	**19.23%**
Both	**03**	**11.54%**
Deep veins	**0**	**0**

Graph 7: Showing distribution of venous ulcers with reference to system affected



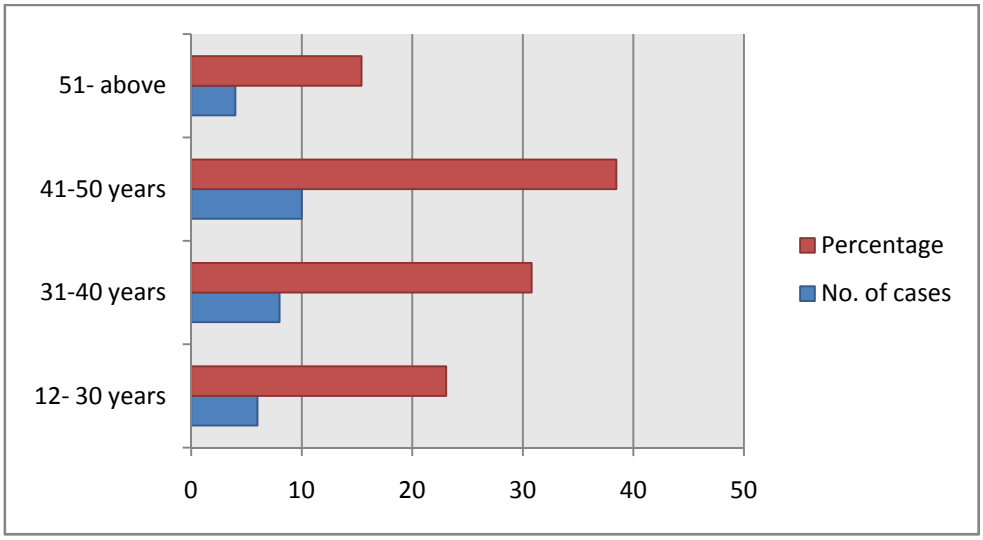
In this study, long saphenous system was found to be by far the commonest system affected in case of venous ulcers accounting for 69.23%.

Table 8

Age distribution of Venous Ulcers

Sl. No	Age group	No. of cases	Percentage
1	12- 30 years	06	23.07%
2	31-40 years	08	30.8%
3	41-50 years	10	38.46%
4	51- above	4	15.4%

Graph 8: Age distribution of venous ulcers



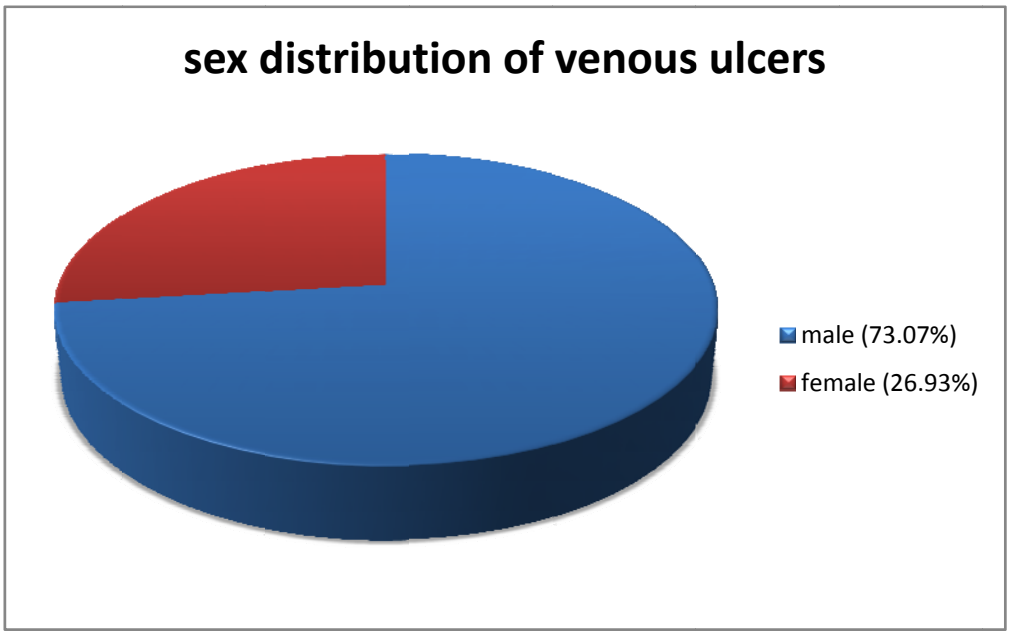
Venous ulcers were found to be the commonest between the age group 31 – 50 years.

Table 9

Sex distribution in Venous Ulcers

Sex	No. of cases	Percentage
Male	**19**	**73.07%**
Female	**07**	**26.93%**

Graph 9: Showing sex distribution of venous ulcers



Males were more commonly affected accounting for 73.07%%. In other published studies it is noted that females have a slightly more preponderance over males.

Arterial Ulcers

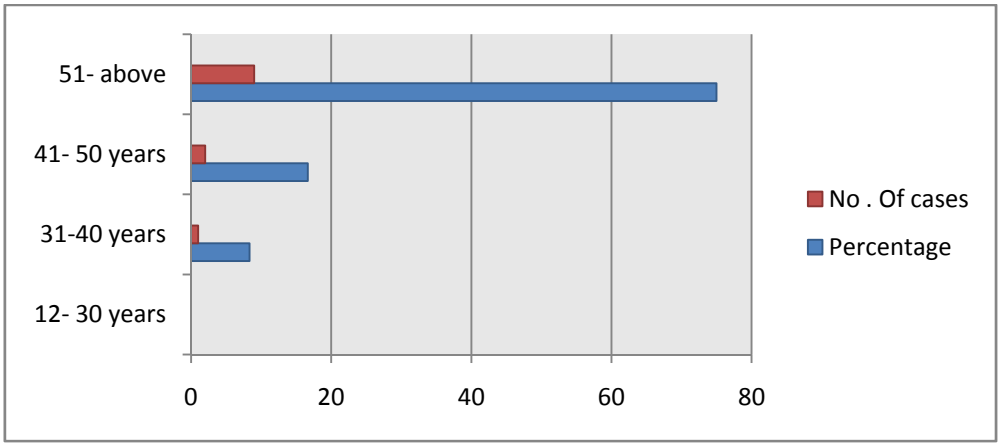
Out of 100 cases, 12 were arterial ulcers.

Table 10

Age distribution of various types of arterial ulcers

Sl.No	Age group	No. of cases	Percentage
1	12- 30 years	0	0
2	31- 40 years	01	8.33%
3	41- 50 years	05	41.66%
4	51 years - above	06	50%

Graph 10: Showing Age distribution of arterial ulcers



Arterial ulcers were found to be the most common ulcers in the age group of 41- 50 and above

Peripheral vascular diseases are 7 times more frequent in 60-year-old when compared to 70years olds according to Hanson Carita

Table 11

Pathology in arterial ulcers

Pathology	No of cases	Percentage
TAO	**5**	**41.66%**
Atherosclerosis	**7**	**58.34%**

Atherosclerosis was found to be the commoner association with arterial ulcers constituting 58.4%. The only other association with arterial ulcers was TAO accounting for 41.6%.

Graph 11: Showing pathology of arterial ulcers

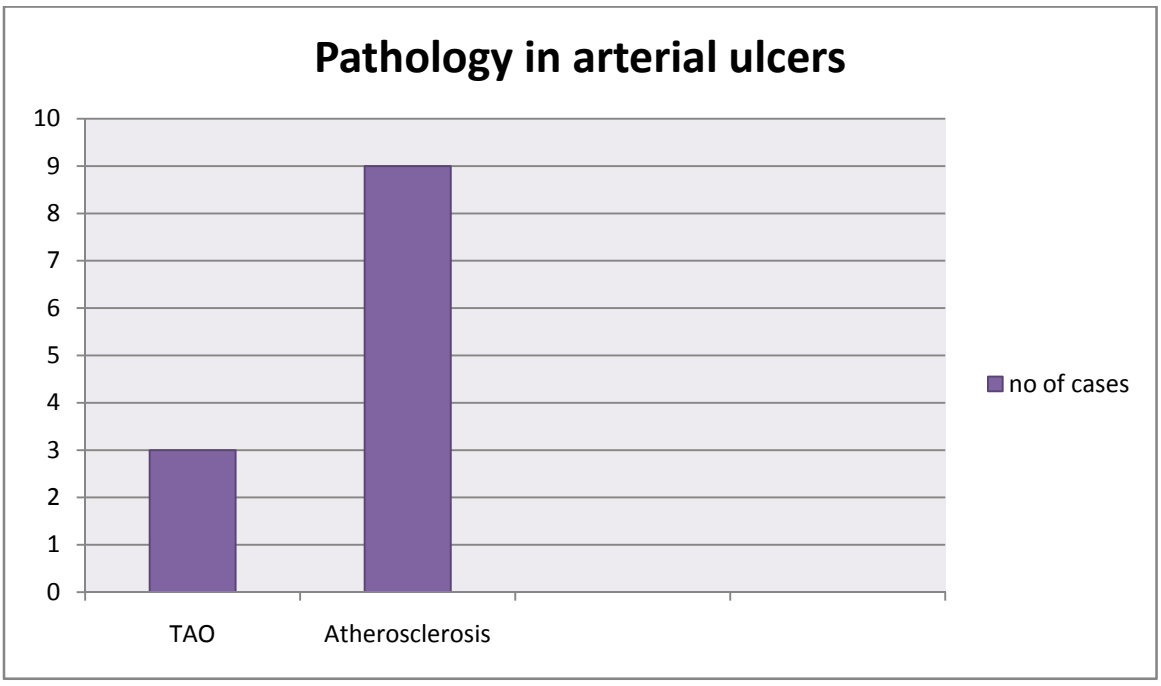


Table 12

Location of the ulcer according to its types

Sl. No	Type of ulcer	Gaiter Zone	Dorsum of foot	Plantar aspect	Total
1	Diabetic	02	28	18	48
2	Venous	23	03	0	26
3	Arterial	0	0	12	12
4	Malignant	0	03	02	05
5	Others	0	01	02	03

The venous ulcers occurred more commonly in the gaiter zone (88.4%). Where as arterial and diabetic ulcers occurred mainly in the toes and foot i.e., 100% and 95.8% respectively. About 60% of malignant ulcers of lower limb occurred in the foot

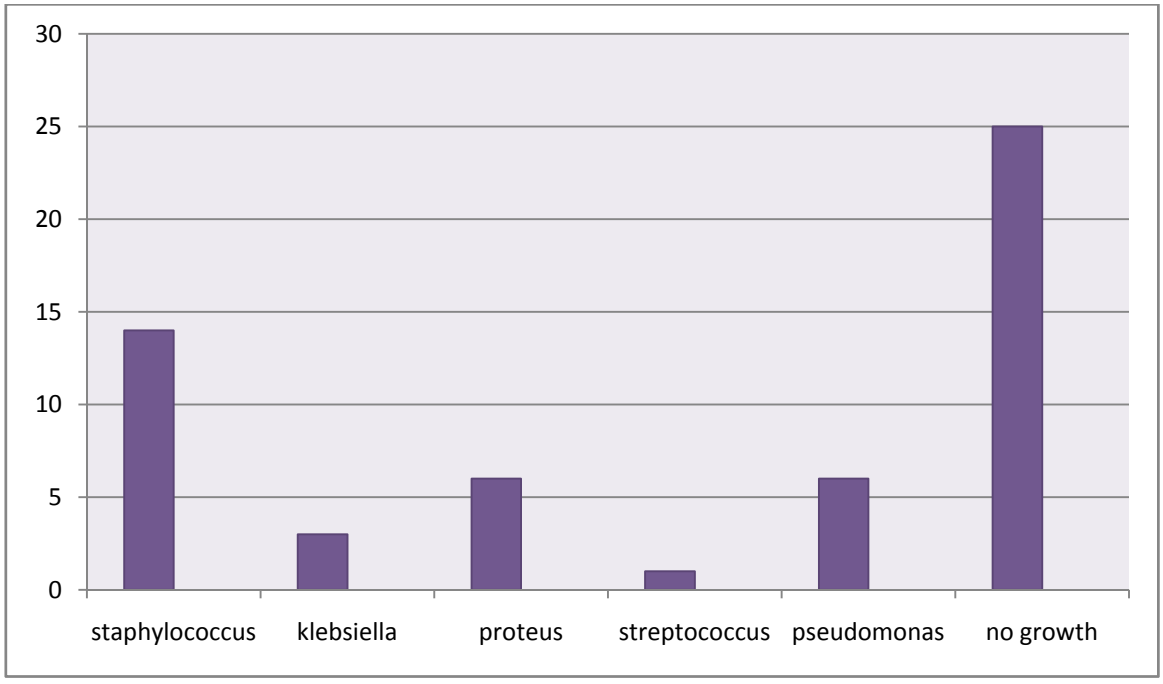
According to Hanson Carita ulcers below the line of shoe and feet are considered mostly caused by arterial insufficiency and or diabetes. Ulcers on the gaiter zone are mostly caused by varicose veins.

Table 13

Types of bacteria isolated from the ulcers

Sl.No	Pathogen	No of cases	Percentage
1	Staphylococcus	14	25.45%
2	Klebsiella	03	5.455%
3	Proteus	06	10.91%
4	Streptococcus	01	1.81%
5	Pseudomonas	06	10.91%
6	No growth	25	45.45%

Graph 12: Showing types of bacteria isolated from the ulcers



Only 55 cases were sent for culture and sensitivity tests. Staphylococcus was found to be the most common pathogen accounting for

25.45% of the bacteriological isolates. This was followed by proteus and pseudomonas, which accounted for 10.9% each, Klebsiella which accounted for 5.45%, streptococcus accounting for 1.81%.

Staphylococcal infection is the most common infection is diabetic foot.

Most of the patients in this study group belong to the lower socio-economic status.

DISCUSSION

The prevalence of foot ulcers is probably between 0.18% and 1% (Phillips, Tania et al).**5** 95% of lowerlimb ulcers are due to vascular etiology, (Gilliland)**7** and among all chronic wounds lower extremity venous ulcer dominates the differential diagnosis accounting for up to 90% of the cases (Burton S. Claude)**8** (Callum M. J. et al).**9** Arterial diseases account for 5% to 10%, most others are due to neuropathy or a combination of both (Yound J. R).**10**

In this study chronic ulcer with vascular etiology accounted for only 38% of all chronic ulcers. Out of this venous ulcers accounted for 26% and arterial ulcers accounted for 12%. Chronic ulcers associated with diabetes accounted for nearly 48%. Malignant ulcers accounted for 5% and other ulcers for 6%.

As observed above the present study was not comparable with the published studies mentioned probably because of following reasons:-

- The study group of 100 patients only hence difficult to draw any comparative conclusions.
- The other published studies were population based, controlled randomized or a group-based study which included different specialties where as this study was a nonrandomized and uncontrolled study.

Some investigators have classified diabetic ulcers as metabolic. The most important factors responsible for causation of ulcer in diabetes are the arterio-sclerotic lesions in large leg arteries and or neuropathy resulting in decreased sensation. If diabetic ulcers in our study are considered vascular disorders rather than metabolic, the percentage of vascular ulcers in our study is about 76% - somewhat comparable to the above study. However, this is controversial and in diabetes it is a combination of factors that are to be considered in causation of leg ulcers.

Also according to Yound J. R.**10** and Boyd A. M. et al,**30** the distribution of different type of ulcers in different studies varies – 70% to 90% for venous ulcer, 5% to 15% for arterial ulcers and 1% to 5% for other ulcers.

As per studies done by Hansson Carita**11** on leg and foot ulcers, ulcers below the line of shoe and feet are considered mostly to be caused by arterial insufficiency and or diabetes. Ulcers on the medial aspect of the ankle in the gaiter zone are mostly caused by venous insufficiency.

In the present study, ulcers had the same site of distribution i.e., ulcers in the gaiter zone were mostly caused by venous insufficiency and ulcers in the foot below the line of shoes were mostly caused by arterial insufficiency and or diabetes.

About 41% of patients in our study had ulcers in the foot only. This is rather high figure in comparison to Hansson's study which showed about only 30% of the ulcers in the foot. This is probably due to more number of diabetic and arterial ulcers in our study.

Cornwall et al**31** in his study had 70% of patients over the age of 70 years. The median age of all patients in this study was 45 years and 44% of the patients over the age of 45 years and had 70% of the patient over the age of 70 years. But according to study done by Callam M. J.**9** the elderly are not the only population at risk: In his study ulceration began before the age of 40 years in 22% of the population studied. In our study, ulceration began before the age of 40 years in 15%% of the patients.

Peripheral vascular diseases increase with age and are 7 times more frequent in 60 years old patients when compared to 20 years old. (Hansson Carita).**11** In this study, arterial and venous diseases were found to be maximum in the age group of 31 to 50 years. This discrepancy may be due to the fact that, our study group patients in the above age group belong to the working class and the ulcers they suffer from hamper their working capacity making them seek medical help early. And also venous ulcers were found to be most common in the age group of 31 to 50 years which is rather early when compared to western studies as most of our patients belong to the working class which involved long hours of standing.

Arterial were found to be more common in the age group of 31 to 50 years which again is rather too early as compared to western studies, since we have in our study a significantly high number of TAO cases which are common in young adults.

In our study, there were more men 65% than women 35% with foot ulcers. However, no differences between sexes were found when age specific relative frequencies for all ulcers were compared.

Elastic crepe bandages are the most important forms of treatment for venous ulcer patients (Rightor M. Myers M. B).**32** In our study all the 26 patients who had venous ulcers wore for elastic crepe bandages stretched to 50% providing of around 14 mmHg compression pressure under one layer. These patients were also subjected to local dressings and Bisgaard's line of management. Once the ulcers healed they were taken up for surgery. Out of the 26 patients, 26 were due to varicose veins. Out of 26 patients with varicose veins, 14 underwent surgery in form of ligation and or Trendelenburg's operation and sub fascial ligation. The mean time for ulcer healing was 17.2 days.The patient who underwent skin graft had his ulcer healed in 7 days only.

A study of recurrences of venous ulcers could not be made due to inadequate time follow up.

Appropriate anti-diabetic therapy informs of plain insulin (Bovine), antibiotics, the debridement and regular dressings were the important methods of treatment for diabetic ulcers in our study. Out of the 48 patients, 40 patients were managed with regular dressings; antibiotics slough excision and or debridement along with anti-diabetic therapy. 3 patients underwent amputation as a life saving measure and one patient expired due to Medical causes. 4 patient underwent skin grafting and had his ulcer healed in 10 days. However, +the mean healing time was 26.43 days in overall diabetic ulcers.

Skin is the best dressing (Lister). It can be applied as a partial thickness graft or numerous pinch grafts. It is best reserved for large ulcers or those, which will not heal, by conservative management (Gilland E. L., John H. N. Wolf).7

OBSERVATION

- The highest age incidence of foot ulcers in this study was in the age group of 51years and above (69%).
- The median age was 45 years and the mean age was 44.28 years.
- There was a marked male predominance of 65%
- Foot- dorsum and toes were the most commonly affected region 88%
- 87.5% of venous ulcers were situated in the gaiter zone.
- 88% of diabetic ulcers were situated in the foot.
- 60% of the arterial ulcers were situated in the foot.
- Of malignant and other ulcers 60% were situated in the foot and 40% heel.
- Staphylococcus was found to be the most common pathogen to be isolated from the ulcers i.e., 25.45%
- 7 patients with foot ulceration had infective gangrene of deeper tissues and they underwent amputation as a life saving procedure and 2 patients with malignant foot ulceration also underwent amputation.
- Most patients with varicose veins underwent some form of operation i.e., ligation and stripping and or Trendelenburg's operation and sub-fascial ligation following healing of ulcers. No recurrences of ulcers were noted.

SUMMARY

Clinical study of ulcers of the leg was carried out at Tirunelveli Medical College Hospital, Tirunelveli from March 2011 to March 2012. The study reveals certain important data. The highest number of cases was found to be ulcer of the foot associated with diabetes mellitus, ulcer due to venous valve incompetence, ulcers due to arterial occlusion secondary to atherosclerosis and TAO. Tropic ulcers associated with Diabetes Mellitus and other ulcers following snakebite and certain infections like pyoderma, gangrenosa.

Diabetic ulcer being more common in males, more prevalent in patients above the age of 51 years and above, more often seen in left limb, venous ulcer was very commonly seen in patients who were presenting with venous valve incompetence resulting in venous congestion commonly seen saphenofemoral junction incompetence and perforator incompetence predominantly seen in males patients between 31-40 years of age in the gaiter zone. Atherosclerosis was found to be the common cause of arterial ulcer due to arterial occlusion with high cholesterol levels. The only other arterial ulcer was due to TAO predominantly seen in middle-aged individuals only in the foot. Among the other ulcers, a few cases with snakebite resulting in cellulitis and tissue necrosis had extensive skin loss leading to non-healing ulcer. A few were due to sutured wound, which got infected, and tissue necrosis

resulting in skin loss leading to non-healing ulcer. The commonest organisms cultured from the wounds was found to be staphylococcus, streptococcus, pseudomonas and klebsiella.

Though the causative factors are varied, diabetes mellitus and varicose veins were by far the more common factors. Underlying vascular disorders are the main etiological factors for foot ulcers with diabetes forming a major risk factor. Diabetes was the commonest disease associated with foot ulceration.

Thus, the study of various cases of foot ulcers arouses lot of interest and is mind bogging as far as the treatment of these cases are concerned. What with the availability of arsenal of investigation wide range of antibiotics and with ever improving dressing material, there is certainly a great improvement in treatment of foot ulcers. Skin grafting when it becomes a choice for chronic ulcers with wide defects is indeed the right one.

To conclude here is a saying -

" I dressed the wound, but God healed it"

- Ambrose Pare'

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PROFORMA

CaseNo : OP/IPNo :

Name : DOA :

Age : Sex : DOD :

Address : Occupation :

PRESENT HISTORY

Onset : Cough :

Duration : Heamoptysis :

Site : Evening rise of temp :

Number : Difficulty in vision :

Size : Walking bare foot :

Bleeding : Standing long hours :

Examination of Ulcers

Inspection : Site :

Number : Size :

Shape : Surrounding area :

Palpation : Temperature : Tenderness :

Floor : Edge :

Discharge : Colour :

Bleeding : Base :

Mobility : Depth :

EXAMINATION OF REGIONAL LYMPH NODES:

VASCULAR EXAMINATION:

EXAMINATION OF FOR NERVE LESIONS:

SYSTEMATIC EXAMINATION:

Cardiovascular system :

Respiratory system :

Abdominal examination :

INVESTIGATIONS :

Urine Albumin : Sugar :

Micro :

Blood Hb% TC : DC : ESR :

Blood Sugar : Urea:

Creatinine:

Cholesterol : VDRL:

Culture and Sensitivity:

Biopsy :

X- ray chest :

Doppler study :

MANAGEMENT

Conservative

- ❖ Antibiotics

- ❖ Anti-inflammatory analgesics
- ❖ Vasodilators
- ❖ Elevation of affected limb
- ❖ Active and passive exercise of lower limb
- ❖ Alternate day dressing
- ❖ Anti-diabetic therapy

Surgical

- ❖ Surgical debridement
- ❖ Skin grafting
- ❖ Subfascialligation
- ❖ Ligation and stripping of varicose veins
- ❖ Wide excision
- ❖ Lumbar sympathectomy
- ❖ Amputation

KEY TO MASTER CHART

A → Antibiotic

AD → Anti-diabetic

Ai → Anti-inflammatory

C → Compression bandage

D → Dressing

DM → Diabetes mellitus

Deb → debridement

E → Elevation

Ex → Exercises

FBS → Fasting Blood Sugar

Kleb → Klebsiella

LSV → Long Saphenous Vein

Lig → Ligation

Lt → Left

P → Pentoxyphylline

PPBS → Post prandial blood sugar

Prot → Proteus

Pseud → Pseudomonas

Rt → Right

S → Serous

SS → Sero-sanguinous

Staph → Staphylococcus

Strep → Streptococcus

Strip → Stripping

Symp → Sympathectomy

TAO → Thromboangiitis Obliterans

US → Urine sugar

MASTER CHART

Sl. No	Name, Age,Sex, IP No	Ulcer	Duration	Size, shape	No	Edge	Floor	Indurated Base	Discharge	Systemic disease	diagnosis	Pus C & S	Spcl inv	Treatment	Results (healing time)
1	Murugan, 50,M,32583	heel of both foot	3 weeks	5x4 cm,4x3cm Irregularl	2	Sloping	Slough	-	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	20 days,
2	Ponmariyammal, 68 F,32743	Heel Lt. foot	8 weeks	6x4 cm,irregular	1	sloping	slough	-	purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	28 days
3	Seyadu, 55 M,31695	Rt. heel	8 weeks	6x4 cm, ,irregular	1	Sloping	slough	-	purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	27 days
4	Rathinasamy, 62M,124024	Lt. third toe	4 weeks	3x3 cm,irregular	1	sloping	slough	-	purulent	DM	Diabetic foot	staph	RBS,US,FBS,PPBS	A,AD,D,Deb	19 days
5	Ganesan 45 M,30948	Rt. dorsum of foot	1 weeks	3x3 cm,irregular	1	Sloping	slough	-	serous	_	Diabetic foot	_	RBS,US,FBS,PPBS	A,AD,D,Deb	27 days
6	Aadhimoolam 75 M,33384	Lt. sole	6 weeks	5x5 cm,irregular	1	sloping	slough	-	purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	24 days
7	Poomariyammal,60 F,38748	Lt. medial malleolous	6 weeks	3x3 cm,irregular	1	Sloping	slough	-	purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	19 days
8	Leela 50 F,33667	Rt. dorsum	4 weeks	6x6 cm,irregular	1	Sloping	slough	-	purulent	DM	Diabetic foot	Proteus	RBS,US,FBS,PPBS	A,AD,D,Deb	29 days
9	Mariyammal 56 f,33709	Both foot dorsum	5 weeks	2x2 cm,irregular	2	Sloping	slough	-	purulent	DM	Diabetic foot	Staph	RBS,US,FBS,PPBS	A,AD,D,Deb	16 days
10	Iyyankannu 60 M,134043	Rt. heel	8 weeks	4x4 cm,irregular	1	Sloping	slough	-	purulent	DM	Diabetic foot	strepto	RBS,US,FBS,PPBS	A,AD,D,Deb	23 days
11	Sornam 55 M,33898	Rt. great toe	8 weeks	3x3 cm,irregular	1	sloping	slough	-	purulent	DM	Diabetic foot	Proteus	RBS,US,FBS,PPBS	A,AD,D,Deb	30 days
12	Samuel 70 M,32386	Lt. dorsum of foot	8 weeks	8x8 cm,irregular	1	Sloping	slough	-	purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	50 days
13	Subbulakshmi 55 F ,124346	Rt. great toe	6 months	4x4 cm, ,irregular	1	Sloping	slough	-	purulent	DM	Diabetic foot	Klebsiella	RBS,US,FBS,PPBS	A,AD,D,Deb	14 days
14	Sornam 51F,204214	Rt. great toe and 2nd toe	3 months	6x3 cm,5x2 cm, ,oval	2	Punched out	slough	-	purulent	MI	TAO	Nil	Doppler Study	D,A, Ai, P, symp	28 days
15	Ulagmadtha 62 F,34290	Lt. sole	3 weeks	3x3 cm,irregular	1	sloping	slough	-	purulent	DM	Diabetic foot	proteus	RBS,US,FBS,PPBS	A,AD,D,Deb	22 days
16	Shanmugavel 68 M,124047	Lt. great toe	6 weeks	1x1 cm,round		Punched out	slough	-	purulent	_	Trophic ulcer	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	14 days
17	Murugesan 50 M,124134	Lt. second toe	1 weeks	1x1 cm,irregular	1	sloping	slough	-	purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	15 days
18	Saraswathy 52 F,35245	Rt. 4 th ,5 th toe	2 weeks	3x3 cm,2x2 cm,oval	2	Punched out	slough	-	serous		TAO	_	Doppler Study	D,A, Ai, P, symp	10 days
19	Arumugam 55 M,35861	Rt. 4th,5 th toe	4 months	1x2 cm, 1x1 cm,irregular	2	Punched out	slough	-	purulent	old MI	Atherosclerosis	_	Doppler Study	Amputation, A,AD,D,Deb	24 days

MASTER CHART

Sl. No	Name, Age,Sex, IP No	Ulcer	Duration	Size, shape	No	Edge	Floor	Induration	Discharge	Systemic disease	diagnosis	Pus C & S	Spcl inv	Treatment	Results (healing time)
20	Sundaravalli 33 F,35950	Rt. medial malleolus	6 weeks	3x2cm Oval	1	Sloping	Slough	_	purulent	_	Venous ulcer	_	Doppler study	E,C,D,Ai, Lig, stripping	12 days
21	Amudha 45 F,35173	Lt. foot	1 weeks	2x2cm	1	Sloping	Slough	_	Purulent	DM	Cellulitis left foot	_	RBS,US,FBS,PPBS	A,AD,D,Deb	15 days
22	Rajammal 64 F,35982	Rt. lateral malleolus	3 weeks	2x2 cm Oval	1	Sloping	Slough	_	Purulent	_	varicose ulcer	_	Doppler study	E,C,D,Ai, Lig, stripping	18 days
23	Chella durai 65 M,243144	Lt. medial malleolous	3 weeks	2x2 cm Oval	1	Sloping	Slough	_	Purulent	_	varicose ulcer	_	Doppler study	E,C,D,Ai, Lig, stripping	21 days
24	Mookan 70 m,36749	Rt. medial malleolus	6 weeks	3x2 cm Oval	1	Sloping	Slough	_	Purulent	_	venous ulcer	--	Doppler study	E,C,D,Ai, Lig, stripping	16 days
25	Sankaran 52 M,242288	Rt. lateral malleolus	3 weeks	2x2 cm Oval	1	Sloping	Slough	_	Purulent	_	varicose ulcer	--	Doppler study	E,C,D,Ai, Lig, stripping	16 days
26	Malaipillai 63 M,242427	Lt. medial malleolus	8 weeks	4x2 cm Oval	1	Sloping	Slough	_	Purulent	_	venous ulcer	--	Doppler study	E,C,D,Ai, Lig, stripping	25 days
27	Madasamy 45 M,242887	Lt. foot	6 weeks	3x2 cm Oval	1	Sloping	Slough	_	Purulent	_	varicose ulcer	--	Doppler study	E,C,D,Ai, Lig, stripping	18 days
28	Krishnan 56 m,37940	Rt. Foot	4 weeks	6x4 cm Oval	1	Sloping	Slough	_	Purulent	_	varicose ulcer	--	Doppler study	E,C,D,Ai, Lig, stripping	29 days
29	Arumugam 67 m,38278	Lt. ankle	6 weeks	4x2 cm Oval	1	Sloping	Slough	_	Purulent	_	varicose ulcer	--	Doppler study	E,C,D,Ai, Lig, stripping	16 days
30	Karupasamy 60 m,38476	Rt. lateral malleolus	4 weeks	4x2 cm Oval	1	Sloping	Slough	_	Purulent	_	varicose ulcer	_	Doppler study	E,C,D,Ai, Lig, stripping	25 days
31	Subaiah 55 m,38862	Rt. 3rd toe	1 weeks	1x1 cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	24 days
32	Peter 50 M,242427	B/l great toe	3 weeks	3x3 ,2x3 irregular	2	Sloping	Slough	_	Purulent	DM	diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	19 days
33	Subramaniyam 50 M,39357	Lt. great toe	3 weeks	2x2cm Irregular	1	Sloping	Slough	_	Purulent	DM	diabetic foot	prot	RBS,US,FBS,PPBS	A,AD,D,Deb	30 days
34	Nathan 65 M,41207	Rt. 3rd toe	2 weeks	1x1cm Irregular	1	Sloping	Slough	_	Purulent	DM	diabetic foot	Staph	RBS,US,FBS,PPBS	A,AD,D,Deb	26 days
35	Muthuramalingam64 M,42428	Lt. great toe	3 months	5x3cm	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Staph	RBS,US,FBS,PPBS	A,AD,D,Deb	23 days
36	Selvamani 48 M,42602	Rt. forefoot	6 weeks	6x7cm	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Pseudo	RBS,US,FBS,PPBS	A,AD,D,Deb	21 days
37	Chelladurai 45 M,42602	Lt. side 4th & 5th toe	10	3x2cm, 2x3cm, Oval	2	Punched out	Slough	_	Purulent	_	TAO	Pseudo	Doppler study	D,A, Ai, P, symp	25 days
38	Selvam 32 M,43568	Lt. heel	2	3x3cm irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Strepto	RBS,US,FBS,PPBS	A,AD,D,Deb	14 days

MASTER CHART

Sl. No	Name,Age,Sex, IP No	Ulcer	Duration	Size, shape	N0	Edge	Floor	Indurated Base	Discharge	Systemic disease	diagnosis	Pus C & S	Spcl inv	Treatment	Results (healing time)
39	Muthusamy 52 M,17221	Rt. heel	3 weeks	2x2cm Oval	1	Punched out	Pale granulation	+	purulent	DM	Trophic ulcer	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	23 days
40	Karuppaya 55 M,44942	Rt. lateral Malleolus	3 weeks	2x2cm Oval	1	Sloping	Pale granulation	_	serous	_	Varicose ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	12 days
41	Esakkimuthu 61 M,46511	B/l great toe	3 weeks	3x4cm 2x2cm	2	Sloping	slough	_	Purulent	DM	Diabetic foot	Pseudo	RBS,US,FBS,PPBS	A,AD,D,Deb	23 days
42	Ganesh kumar 18 M,47989	Rt. medial mallolus	6 weeks	6x4cm Oval	1	Sloping	Pale granulation	_	Serous	_	Varicose ulcer	_	Doppler	E,C,D,Ai,Lig, stripping,	19 days
43	Gandhi 61 F,48398	Rt. heel	2 weeks	4x4cm irregular	1	Sloping		_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	15 days
44	Subramani 80 M,49032	Rt. medial malleolus	6 weeks	3x2cm Oval	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	12 days
45	Murugaiya 65 M,49333	Rt. medial malleolus	6 weeks	3x2cm Oval	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	17 days
46	Sunderson 81 M,48019	Lt. lateral malleolus	6 weeks	4x2cm oval	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	26 days
47	Kannammal 40 F,48020	Rt. medial malleolus	4 weeks	2x2cm oval	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	16 days
48	Ayyakannu 63 M,47304	Lt. lateral malleolus	4 weeks	6x4cm oval	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping, Skin grafting	25 days
49	Rahmath beevi 60 F,49012	Rt. medial malleolus	6 weeks	3x2cm Oval	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	14 days
50	Ramar 38 M,38212	Rt dorsum foot	3 years	6x6cm Irregular	1	Everted	Slough	+	Serous	_	Squamous Cell Carcinoma	_	Biopsy	Excision , grafting	29 days
51	Alagammal 65 F,124345	Rt little toe	3 weeks	2x1cm Irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Staph	RBS,US,FBS,PPBS	A,AD,D,Deb	23 days
52	Ramasamy 36 M,50601	Rt great toe	6 weeks	1x1cm Round	1	Punched Out	granulation	_	Purulent	_	Trophic Ulcer	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	12 days
53	Gnanaprakasham 62 M,51075	Rt great toe	3 weeks	2x2cm Irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS,PPBS	A,AD,D,Deb	19 days
54	Velapandi 45 M,51233	Rt 2^{nd} & 3^{rd} toe	6 weeks	2x2cm 2x2cm	2	Punched out	slough	_	purulent	_	TAO	Pseudo	Doppler	D,A, Ai, P,	12 days
55	Kalathiyam 28 M,53153	Rt medial malleolus	6 weeks	3x2cm	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	16 days
56	Rajasingh 55 M,53291	Rt medial malleolus	6 weeks	6x4cm Oval	1	Sloping	granulation	_	Serous	_	Venous Ulcer	_	Doppler	E,C,D,Ai,Lig, stripping, skin graft	29 days
57	Mariammal 23 F,53963	Rt medial malleolus	7 weeks	3x2cm Oval	1	Sloping	granulation	_	Serous	_	Venous ulcer	_	Doppler	E,C,D,Ai,Lig, stripping	17 days

MASTER CHART

Sl. No	Name, Age,Sex, IP No	Ulcer	Duration	Size, shape	No	Edge	Floor	Indurated Base	Discharge	Systemic disease	diagnosis	Pus C & S	Spcl inv	Treatment	Results (healing time)
58	Parameswari 28 F,53827	Rt medial malleolus	9 weeks	4x5cm oval	1	Sloping	Healthy granulation	_	serous	_	Venous ulcer	_	Doppler study	Skin Grafting	21 days
59	Meena 50 F,54499	Rt dorsum of foot	3 years	6x6cm Irregular	1	Everted	Pale granulation	+	Serous	_	Squamous cell carcinoma	_	Biopsy	D,Ai,Excision , grafting,	29 days
60	Hussain 65 M,55083	Rt forefoot	1 year	8x7cm Irregular	1	Everted, growth	Slough	_	Serous	_	Melanoma	_	Biopsy	Amputation, chemotherapy	23 days
61	Kasiammal 70 F,53581	Both heel	3 weeks	1x1cm, 2x1cm Irregular	2	Punched Out	Pale granulation	_	Serous	_	bedsore	_	_	A, D, debridement	25 days
62	Ramakrishnan 65 M,55774	Rt sole of foot	2 years	5x5cm, Irregular	1	Everted, growth	Slough	_	Serous	_	melanoma	_	Biopsy	Amputation, chemotherapy	24 days
63	Kajamydeen 63 M,55812	Rt dorsum of foot	2 years	5x4cm Irregular	1	Everted, Growth	Slough	_	Serous	_	Melanoma	_	Biopsy	Amputation, chemotherapy	20 days
64	Saraswathi 60 F,54516	Heel of both foot	3 weeks	1x1cm, 2x2 cm Irregular	2	Punched out	Pale granulation	_	Serous	_	bedsore	_	_	A, D, debridement	12 days
65	Paramasivan 28 M,242421	Rt medial malleolus	6 weeks	3x3cm Oval	1	Sloping	Pale granulation	_	Serous	_	Venous ulcer	_	Doppler Study	E,C,D,Ai,	15 days
66	Sankili 60 M,55202	Lt medial malleolous	3 weeks	2x2cm Oval	1	Sloping	Pale granulation	_	Serous	_	Varicose ulcer	_	Doppler Study	E,C,D,Ai,	09 days
67	Gomathi 25 F,242420	Rt medial malleolous	10 weeks	3x4 cm Oval	1	Sloping	Pale Granulation	_	Serous	_	Venous ulcer	_	Doppler Study	E,C,D,Ai,Lig, stripping	17 days
68	Mahalingam 25 M,56415	Lt medial malleolus	10 weeks	3x3 cm Oval	1	sloping	Pale granulation	_	Serous	_	Venous ulcer	_	Doppler Study	E,C,D,Ai,Lig, stripping	15 days
69	Indirani 55 F,56516	Rt dorsum of foot	6 weeks	2x2 cm Irregular	1	sloping	Slough	_	Serous	_	Mycotic ulcer	_	Biopsy	A, D,Ai, debridement	25 days
70	Mayil 53 F,56933	Rt dorsum of foot	1 weeks	4x3 cm Irregular	1	sloping	Pale Granulation	_	Serous	_	cellulitis	_	_	A,AD,D,Deb	17 days
71	Gomathi 70 F,56359	Rt great toe	3 weeks	1x1 cm Irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS ,PPBS	A,AD,D,Deb	23 days
72	Gurusamy 65 M,54571	Rt great and little toe	3 weeks	2x2cm, 1x1cm Irregular	2	sloping	Slough	_	Purulent	DM	Diabetic foot	Pseudo	RBS,US,FBS ,PPBS	A,AD,D,Deb	21 days
73	Iyyadurai 70 M,51787	Lt heel	3 weeks	3x3 cm Irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS ,PPBS	A,AD,D,Deb	16 days
74	Ramaiah 60 M,242470	Lt great toe	3 weeks	2x1 cm Irregular	1	sloping	Slough	_	Purulent	DM	diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	11 days
75	Ramalakshmi 71 F,57515	Lt heel	6 weeks	3x5 cm Irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	18 days

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76	Selvaraj 70 M,57524	Rt little toe	1 week	1x1 cm Irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	No growth	RBS,US,FBS ,PPBS	A,AD,D,Deb	21 days
77	Sahira banu 57 F,57764	Rt great toe	3 weeks	3x3cm, irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	No growth	RBS,US,FBS ,PPBS	A,AD,D,Deb	21 days
78	Parvathi 46 F,58019	Both heel	5 weeks	3x4cm, 4x5cm, irregular	2	Sloping	Slough	_	Purulent	DM	Diabetic foot	pseudo	RBS,US,FBS ,PPBS	A,AD,D,Deb	27 days
79	Rasaiah 50 M, 242427	Rt little toe	1 week	1x1cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	pseudo	RBS,US,FBS ,PPBS	A,AD,D,Deb	09 days
80	Asaithambi 59 M, 242247	Lt great toe	3 weeks	3x3cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS ,PPBS	A,AD,D,Deb	11 days
81	Nambi 70 M,58413	Rt 2nd toe	3 weeks	1x2cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	pseudo	RBS,US,FBS ,PPBS	A,AD,D,Deb	13 days
82	Vinayagam 40 M, 242248	Lt 1st , 2nd toe	6 weeks	2x2cm, 1x1cm, irregular	2	Sloping	Slough	_	Purulent	DM	Diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	22 days
83	Perumal 67 M,58801	Rt great toe	3 weeks	3x3cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS ,PPBS	A,AD,D,Deb	31 days
84	Muthammal 60 F,59515	Rt great toe	1 month	3x3cm, irregular	1	Punched out	Slough	+	Purulent	_	Atherosclerosis	Pseudo	Doppler study	D,A, Ai, P,	16 days
85	Paramasivan 38 M, 247242	Rt 4th , 5th toe	2 weeks	2x2cm 1x1cm, irregular	2	Punched out	Slough	_	Serous	_	TAO	_	Doppler study	D,A, Ai, P,	12 days
86	Paulraj 60 M,54240	Rt 1st 2nd toe	2 months	2x2cm 1x2cm, irregular	2	Punched out	Slough	_	Purulent	_	Atherosclerosis	Nil	Doppler study	D,A, Ai, P,	23 days
87	Karuppayee 70 F,59515	Rt 4th , 5th toe	3 months	4x3cm 3x2cm, irregular	2	Punched out	Slough	_	Purulent	_	Atherosclerosis	Nil	Doppler study	D,A, Ai, P,	27 days
88	Indhurani 55 F,56516	Lt 4th , 5th toe	10 weeks	2x2cm 1x2cm, irregular	2	Punched out	Slough	+	Purulent	_	TAO	Nil	Doppler study	D,A, Ai, P,	31 days
89	Chithirai vadivu 60 F,58790	Rt 2nd , 3rd toe	6 weeks	3x2cm 1x2cm, irregular	2	Punched out	Slough	+	Serous	_	TAO	_	Doppler study	D,A, Ai, P,	15 days
90	Petchiammal 52 F,59390	Rt great toe	2 months	4x4cm, irregular	1	Punched out	Slough	_	Purulent	_	Atherosclerosis	Nil	Doppler study	D,A, Ai, P,	20 days
91	Vasantha 60 F,59293	Rt great toe	1 week	1x1cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS ,PPBS	A,AD,D,Deb	23 days

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92	Kasi 50 M,60742	Lt great toe	4 weeks	4x2cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	27 days
93	Mandhiresan 85 M,60476	Rt lateral malleolus	6 weeks	1x1cm 3x3cm, irregular	2	Sloping	Slough	_	Purulent	DM	Diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	23 days
94	Ramasamy 57 M, 664487	Rt foot dorsum	3 months	6x6cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Proteus	RBS,US,FBS ,PPBS	A,AD,D,Deb	30 days
95	Sivan 38 M, 124149	Lt great toe	8 weeks	3x3cm, irregular	1	Sloping	Slough	_	Purulent	DM	Diabetic foot	Strepto	RBS,US,FBS ,PPBS	A,AD,D,Deb	21 days
Sl. No	Name,Age,Sex, IP No	Ulcer	Duration	Size, shape	No	Edge	Floor	Indurated Base	Discharge	Systemic disease	diagnosis	Pus C & S	Spcl inv	Treatment	Results (healting time)
96	Karuppaiah 70 M,593207	Lt 5th toe	10 weeks	2x2cm, irregular	1	sloping	slough	_	purulent	DM	Diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	20 days
97	Sankaran 65 M,593141	Rt 2nd toe	3 months	2x2cm, Irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	Proteus	RBS,US,FBS ,PPBS	A,AD,D,Deb	19 days
98	Aseervatham 80 M, 593207	Lt great toe	4 weeks	4x3cm, Irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	20 days
99	Karuppaiah 70 M,593016	Rt foot dorsum	4 months	8x8cm, Irregular	1	sloping	Slough	_	Purulent	DM	Diabetic foot	Nil	RBS,US,FBS ,PPBS	A,AD,D,Deb	35 days
100	Petchiammal 52 F,593906	Lt 3rd toe	8 weeks	2x2cm, irregular	1	sloping	slough	_	purulent	DM	Diabetic foot	Staph	RBS,US,FBS ,PPBS	A,AD,D,Deb	25 days